Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.

BULLETIN No. 10.

U. S. DEPARTMENT OF AGRICULTURE.

BUREAU OF ANIMAL INDUSTRY.

CORNSTALK DISEASE,

AND

RABIES IN CATTLE.

AN INVESTIGATION INTO THE NATURE, CAUSE, AND MEANS OF PREVENTING THE CORNSTALK DISEASE (TOXÆMIA MAIDIS) OF CATTLE. By VERANUS A. MOORE, B. S., M. D.

A DISEASE IN CATTLE NOT DISTINGUISHABLE FROM RABIES. By VERANUS A. MOORE, B. S., M. D.

CHEMICAL EXAMINATION OF CORNSTALKS PRESUMABLY THE CAUSE OF CORNSTALK DISEASE IN CATTLE. By E. A. DE SCHWEINITZ, M. D., PH. D.

Investigations conducted under the direction of
DR. D. E. SALMON,
Chief of the Bureau of Animal Industry.

ISSUED JANUARY, 1896.

WASHINGTON:
GOVERNMENT PRINTING OFFICE,
1896.

LETTER OF TRANSMITTAL.

U. S. Department of Agriculture,
Bureau of Animal Industry,
Washington, D. C., November 22, 1895.

Sir: I have the honor to transmit herewith for publication a report on the cornstalk disease of cattle, and also one covering investigations of a disease which is apparently identical with rabies. The cornstalk disease has brought heavy losses to the cattle feeders of the Western States; its causation has been an unsolved mystery, and much that has been written about it has been incorrect, misleading, and injurious to the interests of the cattle owners and of the country at large.

The investigations of Dr. Moore are given in detail in this report, and have been conducted with great care and with all the facilities that modern science has placed at our disposal. It is shown conclusively that the cornstalk disease is not a disease of the lungs; that it is not a communicable malady; and that it is not even a bacterial disease.

In the past a great deal of discredit has been brought upon American cattle by statements that the cornstalk disease is an infectious disease; that it is caused by pathogenic bacteria, which might be inoculated from animal to animal, and that the disease is identical with the broncho-pneumonia sometimes found in cattle after ocean transit. There have never been made any observations or experiments which gave reasonable justification for such conclusions.

So far as is known the cornstalk disease only affects cattle which have fed upon cornstalks as they stand in the fields. It comes on suddenly, most of the affected animals die within a short time, and post-mortem examination reveals no signs of pneumonia or broncho-pneumonia.

It has never been shown that the American cattle found affected with broncho-pneumonia in Europe had ever eaten cornstalks in the fields, and certainly they could not have fed in that way for three weeks prior to slaughter. Nor is there any evidence that any cattle have ever suffered in this country from cornstalk disease two or three weeks after they ceased eating the standing cornstalks. This hypothesis, therefore, could not possibly be made to conform with the known facts at the time it was promulgated, and the investigations subsequently made have entirely disproved it.

The report under consideration indicates the real nature of this obscure disease and the means by which it may be avoided. The prevention requires some change in the methods of feeding, but these changes are in the direction of better and more economical farming, and they will tend to increase the income of the farm even if the losses from this disease are not considered.

The chemical examinations by Dr. de Schweinitz of cornstalks from fields where the disease occurred are summarized in his report, and appear to confirm the conclusion reached by Dr. Moore.

The discovery of a disease affecting cattle which is very closely related to, if not identical with, rabies, and which is apparently quite common in certain parts of the West, is extremely interesting, and may lead to the suggestion of appropriate preventive measures. It has not yet been determined whether these cattle were bitten by a rabid animal; but as that is the only known cause of rabies it is probable that further inquiry will show that this mysterious disease arises from the bites of dogs or perhaps of some wild animals that are affected with it.

Very respectfully,

D. E. SALMON,
Chief of the Bureau of Animal Industry.

Hon. J. STERLING MORTON,
Secretary of Agriculture.

LETTER OF SUBMITTAL.

U. S. Department of Agriculture,
Bureau of Animal Industry,
Washington, D. C., July 30, 1895.

Sir: I have the honor to submit herewith a report on the cause, nature, and means of prevention of the cornstalk disease of cattle; also the results of an inquiry into an outbreak of apparently sporadic rabies. The investigations upon which it is based were begun, in accordance with your instructions, in the fall of 1892, and continued more or less constantly until the spring of 1894. I found, contrary to reports, that outbreaks of the cornstalk disease were quite uncommon. The infrequency of their appearance and the insidious nature and rapid course of this disease have rendered it impossible, up to the present time, to obtain all the data necessary to make this report complete in every particular. The numerous observations and experiments made in the course of this investigation, however, have given uniform results which will have much influence in eliminating many of the widespread errors concerning the etiology and importance of this disease, and in causing the discontinuance of many troublesome and ineffective methods of prevention now in vogue. The data obtained indicate a ready means of prevention which has the additional advantage of materially increasing the forage value of the cornstalks.

The term "cornstalk disease," by which this affection is popularly designated, refers simply to the conditions under which the deaths occur. The fact does not appear to be generally considered that agencies already recognized and well understood by veterinarians cause a certain percentage of deaths attributed to this disease. On account of this unrestricted meaning it becomes necessary to limit the popular term to the deaths occurring under more clearly defined conditions, and it is therefore desirable to substitute for it another name. As this disease is of a toxic nature the name *Toxæmia maidis* is proposed. However, as the term "cornstalk disease" is the one by which this malady is known it is used to avoid confusion, with the limitations herein specified, in the text of this article.

The omission of much desired information, especially concerning the symptoms, is due to the extreme difficulty in reaching outbreaks in time to make ante-mortem examinations of the affected animals. The many unsuccessful efforts which were made to obtain these data have indicated that the only practical way of procuring them is to enlist the cooperation of veterinarians and investigators residing within the geographical boundaries of the disease who may, perchance, have the opportunity of recording these at present undetermined facts. In order that the results of others, as well as the outcome of these researches, may be at hand to guide further inquiry, a brief résumé of the various theories advanced and results heretofore obtained is prefixed to the detailed account of these investigations.

While engaged in the above-mentioned researches my attention was called to an outbreak of a mysterious affection of cattle in northern Iowa, which was causing the death of many animals, and which it was thought might possibly be similar to the cornstalk disease. It was learned that outbreaks similar to this occurred quite frequently, but up to that time the nature of the disease was a profound mystery. The investigation of this outbreak led to the diagnosis of rabies. This is of much importance, as it suggests the possibility of the occurrence of rabies in cattle, or a disease not distinguishable from it, without the intervention of rabid animals. There is much evidence to indicate a causal relation between the conditions under which the animals were kept and the disease.

In carrying out these investigations I was assisted in much of the field work and in certain of the experiments by Dr. F. L. Kilborne. Special mention of the work performed by him will be made in the text. The thanks of the Bureau are due to the authorities of the experiment stations of the States of Iowa and Illinois for the use of their bacteriological laboratories, and for all other possible assistance in furthering these inquiries. The uniform courtesy extended by the owners of the affected herds and their assistance and cooperation are likewise acknowledged.

Very respectfully,

VERANUS A. MOORE,
Chief of the Division of Animal Pathology.

DR. D. E. SALMON,
Chief of the Bureau of Animal Industry.

TABLE OF CONTENTS.

Page.

An Investigation into the Nature, Cause, and Means of Preventing the Cornstalk Disease (*Toxæmia maidis*) of Cattle. By Veranus A. Moore.......... 9
  Investigations prior to 1892.......... 11
    Investigations by Prof. John Gamgee.......... 11
    Investigations by Dr. F. S. Billings.......... 13
    Investigations at the Illinois State experiment station.......... 15
  A letter of inquiry and a summary of the replies.......... 16
  Special Investigations.......... 18
    Methods.......... 18
    Outbreaks.......... 20
    Cornstalks affected with "corn blight," or the Burrill disease, are free from pathogenic properties.......... 39
    Feeding diseased cornstalks to rabbits.......... 42
    Experiments to ascertain the extent to which cornstalk fields in which cattle have died are dangerous.......... 43
    The bacillus of the Burrill disease of corn.......... 45
      Description of the bacillus.......... 46
    Corn smut (*Ustilago maidis*) not fatal to animals.......... 47
    Animals other than cattle not affected.......... 49
    Chemical analyses.......... 49
  A Discussion of the Nature of the Disease, Based on the Facts Elicited in the Special Investigations.......... 50
    Conditions under which the disease occurred.......... 50
    Symptomatology.......... 53
    Morbid anatomy.......... 55
    Bacteriological examinations.......... 58
    Etiology.......... 59
    Economic importance.......... 62
    Prevention of the diseases.......... 64
A Disease in Cattle not Distinguishable from Rabies. By Veranus A. Moore.. 71
  Symptoms and lesions in the cattle examined.......... 72
  Comparative inoculations.......... 77
  Inoculations with the virus originally obtained from steer No. 4.......... 77
  Inoculations of rabbits and a calf with the brain of a rabid dog.......... 83
Chemical Examination of Cornstalks Presumably the Cause of Cornstalk Disease in Cattle. By E. A. de Schweinitz.......... 88

ILLUSTRATIONS.

To face page—

PLATE I.—Engorgement of Blood in the Liver.......... 66
PLATE II.—Hemorrhages in the Heart Muscle.......... 68

CORNSTALK DISEASE, AND RABIES IN CATTLE.

AN INVESTIGATION INTO THE NATURE, CAUSE, AND MEANS OF PREVENTING THE CORNSTALK DISEASE (TOXÆMIA MAIDIS) OF CATTLE.

By VERANUS A. MOORE.

(Plates I and II.)

The name "cornstalk disease" has been given to a somewhat mysterious affection from which cattle sometimes suffer while feeding in cornstalk fields late in the fall and early winter. The meaning generally accepted and intended to be conveyed by this term is, that an animal or a number of animals, usually cattle, have died suddenly either while feeding in a cornstalk field or very soon afterwards. From a pathological standpoint, therefore, the term is meaningless, but it has served admirably as a generic term to designate certain fatalities which were not easily explained otherwise. Geographically, this affection is restricted to those sections of the United States where the farmers harvest their corn by picking the ears from the standing stalks, after which cattle are turned into the cornstalk fields. At present, therefore, it is limited in its distribution to the middle and northern portions of the Mississippi Valley.

The disease is very insidious, frequently causing the death of the animals before its presence is suspected. Farmers in the affected region frequently observe that their cattle act perfectly well at night, and on the following morning one or more of them are dead. Local veterinarians and butchers, in making autopsies on the dead animals, report the failure to find lesions sufficient to cause death. Usually all the animals in a herd which die, perish in a single night, or, at the longest, within a few days after the first death occurs. It is generally believed to be invariably fatal, and its symptomatology has not been fully determined.

This affection of cattle has been known to exist for many years. The place and time of its origin, however, are not known, but it seems to be peculiar to the United States, and to have first appeared west of the Alleghany Mountains. Farmers tell us of its occurrence in the Mississippi Valley forty or more years ago. They usually refer to its extensive ravages in 1864–65, and again in 1868. From an historical standpoint, no positive statements can be made concerning it prior to 1868, when the first recorded investigations into its nature and cause were made. After that time the disease seems to have escaped the attention of investigators until 1889, when Dr. Frank S. Billings, of the agricultural experiment station of Nebraska, published the results of his investigations. His "germ theory" and the wide distribution of his publications gave to this disease of cattle an unwarranted conspicuousness, and did much to direct attention to the malady, which he set forth as one of vast economic importance. This aroused the fears of cattle raisers, who depended in part or wholly upon corn fodder to winter their animals, and revived both the interest in this disease and the fear of it.

A careful review of the recorded statements shows that many causes have been assigned to it. The various theories have, for the greater part, been formulated by farmers, cattle raisers, and local veterinarians, and, as might be expected, they are based very largely upon the history of a limited number of outbreaks. A more careful inquiry into the origin of these theories shows that as the disease, with occasional exceptions, occurred in only one or two herds in a neighborhood during the same season, the local conditions affecting these herds were supposed to be favorable to the disease, if not its exciting cause. It would be useless to attempt a consideration of all the causes which have thus been suggested. There are, however, certain theories which appear to possess a greater or less degree of probability, and which can not be gainsaid without positive evidence to the contrary.

It is a curious fact that, while nearly every cattle owner in the sections where this disease prevails, in his experience, entertains one of the popular theories respecting its cause, the other supposed causes have proven to be erroneous or for other reasons are believed to be spurious. There is no uniformity of opinion concerning its etiology. The theories most commonly entertained are appended:

(1) A lack of salt and an insufficient supply of water.

(2) Feeding the cornstalks without an admixture of other food, such as hay, straw, grain, or pasture grass.

(3) Impaction of the third stomach (omasum) due to overfeeding upon the dry cornstalks.

(4) The ingestion of corn smut (*Ustilago maidis*).

(5) A species of bacteria which is found in the lesion of a disease of corn known as "corn blight," or the Burrill disease.

The first three of these theories are of popular origin and are not supported by the general experience of cattle raisers, as will subsequently be shown. The "smut theory," which was somewhat strengthened by the report of Professor Gamgee in 1868–69, has many adherents. So firmly do certain farmers believe it to be the causative agent that they collect and draw out of the field all stalks affected with this fungus. The "germ theory," advanced by Billings, is believed in many sections to be the true explanation of the cause of this malady. The stronghold which the last two theories have on the farmers and cattle raisers of the West, and the inconvenience and often expense involved in carrying out the methods of prevention indicated by them, render necessary a brief consideration of the facts upon which these theories are founded, in order that the results of the investigations here recorded may be understood and properly interpreted. As will be shown, our investigations, which would have verified these theories if they were true, fail to confirm the correctness of any of them.

INVESTIGATIONS PRIOR TO 1892.

THE INVESTIGATIONS OF PROF. JOHN GAMGEE.

In 1868 Gamgee was employed by the United States Department of Agriculture to investigate the cornstalk disease of cattle. Although this disease had been known to exist for many years prior to that time, its ravages had not been sufficiently extensive to enlist any active inquiry into its cause or means of prevention. The "smut theory" appears to have been the prevailing one at that time, and consequently Gamgee's report[1] deals almost exclusively with the effect of improperly prepared food, smuts, etc. Corn smut prevailed to an unusual extent in 1868, and in order to test its etiological value he carried out the following experiment:

> Forty-two pounds of smut free from extraneous material were collected and fed to two cows, together with a small quantity of corn meal and as much cut hay as they could eat. The smut was mixed with corn meal and wet for one animal; the other received it in a dry condition. At first the cattle received 3 ounces of the smut twice daily, but later the quantity was increased to 12 ounces daily. The animal that received the wet meal and smut gained in flesh; the other lost in weight. They were apparently perfectly well at the time the experiment was completed.

The only conclusion which seems to be justified from this experiment is the one Gamgee himself drew, viz, "that smut is not a very active poison in combination with wholesome food." Although the experiment with smut failed to demonstrate any etiological value, he entertained the opinion that the smut and moldy corn were the causative agents in the production of the disease.

In his report a somewhat interesting description of the symptoms of this disease is given. Although the symptomatology is quite exhaustive the text is not clear on the point whether or not the symptoms were observed by himself or reported to him by the owners of the cattle. His description of the symptoms and pathological changes in animals affected with this malady is as follows:

> *Symptoms of the ill effects of smutty corn.*—Cattle fed on smutty cornstalks first denote ill health by constipation. It is true that a farmer may be attracted only by an animal lying down, with an unthrifty look, staring coat, dry muzzle, and perhaps

---

[1] Diseases of Cattle in the United States. U. S. Department of Agriculture, 1869, p. 73.

trembling; or a steer may be noticed "tucked up," with hind limbs drawn under, head depressed, shivering, dullness of eyes, and anxious expression of countenance. In the third variety the animal seems excited, breathes quickly, and is apparently somewhat delirious; indeed, in the condition described by Mr. Cumming, of Ellen, Aberdeenshire, resulting from impaction of the third stomach, as in cases of lead poisoning, nothing is more strange than this delirium, associated as it is sometimes with blindness. A farmer writing me from the West says that when he tried to put a rope around the head of a sick cow, which he found standing with all the symptoms of sickness presented by other animals of the herd which had been with her in the cornfields, she turned and fought furiously. I have seen an animal in this condition tied up in a stall, rush forward, plunge, strike against any obstacle, roar, moan, grunt in breathing, and appear to suffer acutely if touched or disturbed. In other words, with the impaction of the third stomach, which is the essential lesion of the disease, whether induced by smutty stalks, old indigestible stalks that have no smut, or other kind of food or poison, there are two distinct conditions induced—the one of stupor, listlessness, vertigo, and depression of spirits, indicated by the animals standing sullenly until they drop or are relieved; the second is a state of exquisite sensitiveness, a hyperæsthesia of the skin and system generally. The animals are not only excited, but in a state of actual suffering, and die very speedily in a state of coma or in convulsions. The disease does not last long. I have seen an animal linger on four or five days, but usually the whole course of the malady is run in twenty-four to forty-eight hours.

An animal first seems to show costiveness, with a dry mucus over the scanty excrement; and, although apparently undisturbed, and even feeding, may be dead in twelve to twenty-four hours.

The diagnosis of the disease at an early period of its manifestations is therefore important, and it rests on the knowledge of the manner in which animals have been treated and fed (as the simultaneous attack of several animals show), and especially on the observations of a fact that I have usually noticed, that the animals which have eaten most ravenously have been the first and most severely affected. Old cattle may sometimes avoid the smutty food, and young animals eat heartily; the latter will be found the only ones to die.

Post-mortem appearances.—The state of torpor of the alimentary canal of animals affected with this disease is indicated on opening the belly and exposing the stomach to view. In the first stomach, or paunch, corn husks and corn are found in a dry condition. Sometimes the rumen is very full, and gas may have become disengaged in it so as to cause great distention, which is relieved by puncture. The contents of the second stomach, or reticulum, are in the same condition as those of the first, though sometimes mixed with some fluid. The third stomach (manyplies or omasum) is firm, distended, and on being opened the food is found caked between folds, with marked impressions of the papillæ or little eminences which stud the mucus membrane. We find in almost all fevers a similar condition of the third stomach, and indeed in healthy animals it is that part of the digestive organs in which the food is most dry and packed preparatory for solution by the gastric juice and intestinal secretions. But there are other lesions associated with this "caking" of the food in the third stomach, in specific diseases, and its existence without these affords evidence of a primary form of impaction, which has received remarkable names, such as "staking," "bound," "fardel bound," etc. The fourth stomach contains but a scanty quantity of greenish, semidigested matter, is usually reddened somewhat diffusely, and the redness increases at times toward the opening of the small intestines.

The intestine, usually replete with somewhat solid and imperfectly digested food, is usually high colored, especially in the fundus of the cæcum and in the large portion of the colon. The rectum is the seat of ramified redness, and a consistent mucus coats its contents.

Persons have reported a peculiar black color of one lung. This is due only to stagnation of the blood after death in the organ nearest the ground, and the same kind of congestion or settling of the blood is apt to pervade other tissues and organs in the side on which the animal has been lying."

THE INVESTIGATIONS OF DR. F. S. BILLINGS.

In 1889 Billings described the cornstalk disease[1] as an "acute extra-organismal septicæmia, due to micro-organisms belonging to the class of ovoid-belted germs, to which variety of disease also belongs the swine plague, southern cattle plague, Wildseuche, hog cholera, and yellow fever in man."

In January and March, 1888, and in February, 1889, he had occasion to examine pieces of tissue brought from cattle that had died while feeding in cornstalk fields. In the first case the tissues were sent in an unsterilized bottle. From the bits of organs which he thus received he invariably isolated a bacillus which he affirms to be the cause of the disease.

The most interesting part of his investigations was the supposed discovery of the identity of the bacillus which he found in the animal tissues with the one described by Burrill[2] as the cause of a disease in cornstalks. This hypothesis is supported by the fact that by feeding the diseased stalks to a rabbit he produced a fatal disease and from the organs of the dead rabbit he obtained pure cultures of the bacillus. He sent a culture of his bacillus to Burrill, who stated that the organism which he (Billings) had obtained from the organs of dead cattle appeared to be identical with the bacillus which produced the disease in the cornstalks. The correspondence between these two investigators, published by Billings, shows that Burrill was conservative on this question, pointing out obvious resemblances only. The responsibility of the statement that the two bacilli are identical rests with Billings.

Dr. Billings found this micro-organism to be fatal to rabbits, guinea pigs, and mice, when inoculated with small quantities of a pure culture. A rabbit fed with a pure culture died in four days and pure cultures of the bacillus were obtained from each of the organs. A calf and a hog were inoculated subcutaneously with 6 c. c. of a pure bouillon culture. The hog remained well, but the calf became visibly affected. The temperature rose to 41.5° C. (106.7° F.). It became very much emaciated, but eventually recovered. Six days after the inoculation he obtained a pure culture of the bacillus from the blood which he drew from a vein at the root of the tail. In a subsequent experiment he inoculated a calf in the jugular vein with 9 c. c. of a pure culture of the bacillus. The calf died in two days. He also produced fatal results in one of two pigs by feeding them the viscera of the calf which died from the intrajugular inoculation. The other pig sickened but recovered. From the dead pig pure cultures of the bacillus were obtained.

---

[1] Bulletin of the Agricultural Experiment Station of Nebraska, Nos. 7, 8, 9, and 10. Original Investigations of Cattle Disease in Nebraska, 1886–1889, University of Nebraska, 1889, p. 164.

[2] Proceedings of the Society for the Promotion of Agricultural Science, 1889, p. 19.

As would be expected, the pathology of the disease which he gives is extremely meager, owing to the fact that all the tissues he saw were brought to him in a few small bottles or jars. In a few cases they are said to have been much changed by decomposition. Nevertheless he found pneumonia to be one of the lesions characteristic of the disease, and in a subsequent bulletin[1] he places great importance upon this lesion, although he records very few additional observations to sustain the claim. In this publication, however, he attempts to show that chronic interstitial pneumonia in cattle is a sequel to the cornstalk disease. The argument is a confusion of statements from various English and French veterinarians which, for the greater part, appeared after the supposed discovery of cornstalk disease in American cattle at La Villette, France. The importance of lung lesions, which he emphasizes in his second bulletin, appears to have been suggested by the following incident, which temporarily gave to this disease an undue importance from a commercial standpoint:

In 1890 a few animals from a shipload of American cattle landed at La Villette, France, died of pneumonia. They were examined very carefully by Nocard and other French veterinarians. From the diseased lung Nocard obtained a microorganism which corresponded very closely to the description of the bacillus of the cornstalk disease of cattle in America, described by Billings. The publication[2] of this fact gave rise to a temporary supposition that this American cornstalk disease might be a menace to the cattle of Europe, and consequently initial steps were taken to require American cattle to be quarantined against it. The fact was subsequently determined that the bacillus isolated by Nocard was that of a form of bovine pneumonia occasionally met with in America, but not known to be contagious, and the matter was dropped.

The symptomatology as described by Billings is confined to the inoculation disease. These symptoms, however, are so confused with those of swine plâgue and Texas fever that they are not of diagnostic value.

In a recent popular publication[3] Billings makes the following statements:

> It is now six years since I first discovered a germ in cattle sick and dying from being turned into stalk fields. In many cases I have carefully examined the whole field, and in some of the worst outbreaks smut was most conspicuous by its almost total absence. Again, Gamgee reports that the manure of his cows, which ate 31 pounds each, was black. In all the autopsies I have made—and they run up to the

---

[1] The Corn-Fodder Disease in Cattle and Other Farm Animals, etc. Bulletin Nos. 22 and 23 of the Agricultural Experiment Station of Nebraska, 1892.

[2] Nocard. Une Broncho-pneumonie infectieuse des Bœufs Américains. The Cornstalk Disease. Recueil de Médecine Vétérinaire. Tome VIII (1891), p. 424.

[3] Breeders' Gazette, Vol. XXVI, 1894, p. 340.

fifties—and among all the sick cattle I have seen there was not an indication of any discoloration of the manure unless it was abnormally light yellow in color. Not only in every case personally examined, but in still more where blood and organs have been sent me, have I isolated one and the same germ, but have repeatedly produced the disease in cattle with inoculations of pure cultures. Some six weeks since I received some corn from a field in which cattle were dying in Indiana, and in this corn I found the same germ. I inoculated rabbits with some small fragments of the diseased corn and it killed them, and from them I made pure cultures and injected 2 cubic centimeters into the jugulars of two cattle (2-year-olds) with the result of killing one and making the other very sick, getting the same germ from both.

So long as these people do not seek a germ in the cattle, so long as they ignore so much proof—and it must be remembered that the same germ has been found and proved in our cattle in France, and the same lesions reproduced by inoculation—so long have these men absolutely no right to advocate the smut theory.

He suggests as a means of prevention that all the diseased cornstalks should be destroyed and animals which die of the disease should be carefully buried or burned. The suggestion is also made that in order to detect the presence of the disease the temperature of the cattle should be taken each night, and all animals which give a temperature over 100° F. must be looked upon as suspicious and those in which it exceeds 102° F. as diseased.[1]

INVESTIGATION AT THE ILLINOIS STATE EXPERIMENT STATION.

A single experiment was made at Champaign, Ill., in 1890, in which the etiological importance of corn smut was tested. A bacteriological examination of the organs from an animal that died in a cornstalk field, supposedly of this disease, was also made by Professor Burrill in 1889. A copy of the station experiment report, which I was told included all of the work which has been done on this disease at that place, was kindly furnished by Professor Morrow, with the privilege of publishing.

Experiment No. 98.—(Investigation of a disease which sometimes affects cattle when turned into cornstalk fields):

Mr. J. M. Craig reports a loss of nine yearlings as follows:

The first animal died two weeks before the cattle were turned into the stalks and while running on clover pasture. This animal was left where it died, and the cattle when turned into the stalk field had access to the pasture, but were believed by Mr. Craig not to have frequented it, and it was not thought that the first death had any connection with those that followed.

Mr. Craig turned twelve head of fattening cattle into a cornfield of 22 acres, which he estimated yielded 65 bushels per acre, on Sunday, probably November 24, 1889, and one steer died the Friday following. The remaining cattle were removed, and on the following Wednesday thirteen "stock" cattle were put in the field. Sunday five died, one died Monday, and one Tuesday night. The remaining cattle were then removed.

December 13, at the request of the experiment station Mr. Craig put the six head of cattle which survived again in the field.

---

[1] It seems strange that such a statement as this should be made when standard German authorities give the normal temperature of cattle from 101° to 101.8° F. Dr. E. C. Schroeder, of this Bureau, who has had much experience in applying the tuberculin test for tuberculosis in cattle, finds that the normal temperature of cattle varies from 101° to 103° F.

December 26, one steer was reported sick. The animal was visited and found to be dull, pulse strong, regular, 56 per minute. Took sample of urine at 6.10 p.m., being the third passage in 30 minutes (Lab. No. 167). Urine afterwards passed as follows: 6.20, 6.25, 6.29, and 6.32.

December 20, three yearling steers were purchased by Mr. Craig and placed in the cornfield.

December 23, a steer was perfectly well the previous evening at sundown, but early in the morning it was unable to get up. At 10.30 a. m. the pulse was about 90, membranes pale, breathing 12 per minute. Dr. McIntosh decided that the animal was unable to live and had him killed. A post-mortem examination was made by Dr. McIntosh and every part pronounced normal except a slight exudation in the liver. Examinations and cultures from spleen, liver, etc., by Burrill failed to reveal any organisms.

The six head that were placed in the field December 13 were removed.

To ascertain whether the smut was responsible for the disease, about 60 pounds of it were collected and fed to a grade Jersey steer, with grain and hay as follows:

From December 13, 1889, to January 2, 1890, 120 pounds of grain, 105 pounds of hay, 20 pounds of smut, and 371 pounds of water were given. From January 2 to January 24, 1890, 176 pounds of grain, 135 pounds of hay, and 39 pounds of smut were fed and 366 pounds of water given. When the experiment was begun December 13, 1889, the steer weighed 560 pounds. January 2, 1890, he weighed 551 pounds, and January 24, 555 pounds. No evidence of disease was discovered.

A LETTER OF INQUIRY AND A SUMMARY OF THE REPLIES.

The absence of reliable data concerning the nature, distribution, and importance of this disease led to the issuing of a circular letter in 1891 to a large number of farmers and cattle raisers by Dr. D. E. Salmon, Chief of the Bureau, for the purpose of obtaining more extended information concerning this disease. Although the replies to the various questions in this letter can not be considered from a scientific point of view altogether accurate, they were given in good faith and indicate the ideas then entertained by interested parties concerning the nature of this disease. During the past few years, however, the "germ theory," as announced by Billings, has become better known through the publication of his results and is more generally accepted as the correct explanation of this mysterious disease. Furthermore, the replies received are important in showing the economic importance attached to this disease by those who suffer most from its ravages. The letter and a summary of the replies are appended:

WASHINGTON, D. C., *March 20, 1891.*

SPECIAL CORRESPONDENT OF THE BUREAU OF ANIMAL INDUSTRY, DEPARTMENT OF AGRICULTURE.

SIR: For many years cattle feeders and stockmen have recognized a disease which has caused extensive losses among their cattle in the fall of the year when the animals were turned into cornfields to feed upon stalks.

Various causes have been assigned for the trouble, such as smut, indigestion from the hard, woody nature of the stalks, and lack of nutriment in the leaves, lack of salt, insufficient supply of water, and a germ producing the so-called "cornstalk disease."

This Bureau contemplates entering upon an investigation into the nature of the malady, but before commencing the critical examination, for which material can not be secured until next fall, we wish to collect information concerning the prevalence, symptoms, and other facts relating to the disease.

Accordingly this circular letter has been addressed to you as a special correspondent of the Bureau of Animal Industry with the hope that your experience will be a valuable aid to us in the investigation.

If those receiving this circular will promptly and as accurately as possible answer the questions on the opposite page they will confer a favor on the Bureau and assist in collecting information which may prove of great importance to the cattle industry of the United States.

A return envelope is inclosed, which can be used without the payment of postage.

Very respectfully,

D. E. SALMON, *Chief of Bureau.*

QUESTIONS.

(1) Have you or your neighbors ever lost cattle from turning them into cornstalk fields?

(2) How many cattle were turned into the fields and how many died?

(3) How were cattle fed before being turned into the cornstalk fields?

(4) How soon after they were turned into the stalk fields did any of the cattle die?

(5) How long before deaths ceased?

(6) What were the general symptoms of the disease?

(7) Did stock receive a liberal supply of salt and pure water?

(8) Did they receive any food in addition to the cornstalks?

(9) Have you noticed that some fields in the same neighborhood are more dangerous than others at the same time of year? If so, was there anything in the situation of the field or condition of the stalks to account for it?

(10) Have you observed any disease of the cornstalks in the fields where the cattle disease was contracted?

(11) Have any horses or other animals died from turning them into cornstalk fields?

(12) Is the disease becoming more frequent than formerly, or is it disappearing?

(13) Do you take any precautions for preventing the disease? If so, what are they, and what is your success?

There were received from the States of Indiana, Illinois, Iowa, Nebraska, Kansas, and Missouri a total of 634 replies to this letter, in which some or all of the questions were answered. Of these, 394 reported that either they or their neighbors lost cattle in cornstalk fields the preceding year. Sixty of these correspondents reported the disease in horses, and curiously enough 32 of them resided in the State of Kansas. Eight reported it in sheep, and 6 of these were from Kansas. The disease was not reported to occur in other animals.

A perusal of the replies to question 3 shows that the great majority of the affected cattle were on pasture land prior to turning them into the cornstalk fields.

The time that elapsed after turning the cattle into the cornstalks until death occurred is reported to be from four hours to two months. Of the 328 answers to the fourth question, 48 of them stated that death occurred in twenty-four hours; 59 in two days; 45 in three days; 23 in four days; 20 in five days; 13 in six days; 47 in seven days; 51 in from one to two weeks; 15 in three weeks; 5 in one month, and 5 in from one to two months. A few others indicated its occurrence in from four to eight hours.

The symptoms given were very numerous. Indigestion, bloating, constipation, "crazy," "stupid," and "dry murrain" were stated to be indications of the presence of the disease.

Fourteen outbreaks were reported to have occurred among cattle which were supplied with salt but no water; 35 outbreaks occurred where there was plenty of water but no salt. In 254 of the outbreaks reported there was an abundant supply of both water and salt, and in 123 outbreaks the cattle received neither salt nor water.

In answer to the question concerning food other than the cornstalks, 302 people replied that no other food was given, while 107 owners fed hay, straw, grain, or allowed the cattle to run at will in pasture or meadow land (usually meadow). The reply to this question was omitted in over 200 of the returned letters.

To the ninth question the answers were equally divided between yes and no. Some men stated that low ground was most dangerous, others that the disease occurred upon high ground most frequently. The presence of smut (*Ustilago maidis*) was the only abnormal condition of the cornstalks reported and was indicated in about one-third of the fields where the disease occurred.

The answers to the question whether the disease is becoming more frequent than formerly, or is disappearing, are interesting. Of those who replied to this question 115 stated that it was common, 50 stated that it was on the increase, while 264 wrote that it was decreasing, and a few others stated that there was no such disease.

Numerous methods for preventing the disease were indicated. Furnishing the animals with an abundance of salt and water and allowing them to remain in the stalk fields for only a short time each day during the first few days appear, however, to be the most common preventive methods. The removal of smut comes next. The fact that the disease appeared where these precautions were taken eliminates the desirability of considering them further than to point out the fact that thus far no efficient means of prevention has been adopted. A few of the customary preventive measures will be considered in connection with the outbreaks investigated.

SPECIAL INVESTIGATIONS.

METHODS.

The investigation into the nature and cause of this disease was attended with many difficulties. The facts that the disease appears at irregular intervals over a large territory, that there is no certainty of its appearing or recurring in any particular locality, that the affected animals are usually found dead, and that frequently all the deaths in an outbreak occur in a single day rendered the adoption of a method for procuring suitable material a matter of considerable difficulty. From the existing knowledge of the disease it was evident that laboratory work was quite as essential as post-mortems for the success of the investigation. The conditions mentioned rendered necessary the adoption of some plan whereby notice of the occurrence of the disease could be received promptly and the affected herd visited in the shortest possible time. A careful consideration of these and other conditions necessary for this investigation led to the conclusion that some place within the infected district should be selected where the necessary laboratory facilities could be obtained, and from which it would be possible to reach the affected animals before it would be too late to make the desired observations.

In carrying out this plan arrangements were made with the authorities of the State experiment station at Ames, Iowa, whereby the use of their bacteriological laboratory was obtained. Further assistance was afforded by the cooperation of Drs. Stalker and Niles, of the veterinary department. Dr. Niles assisted in many of the post-mortems and in the laboratory work. Through the kindness of Professor Wilson, director of the station, a notice of the intended investigations was inserted in nearly all of the county papers and all of the agricultural journals of Iowa, requesting that notice of all outbreaks of this disease should be promptly sent to the State experiment station. About one hundred letters were also sent by the chief of the Bureau of Animal Industry to cattle raisers who had replied to the circular letter of the year before, asking them to report promptly any outbreak of cornstalk disease in their locality, as steps were being taken to inquire into its cause and means of prevention. Through these arrangements it seemed highly probable that the opportunity would be given, in view of the extensive losses previously reported, to make careful observations on a considerable number of affected animals both before and after death.

Notwithstanding these preparations, it was found to be impossible to receive notice of the appearance of the disease and to reach the infected herd before the disease was over and the dead animals in several instances advanced in post-mortem changes. The disease usually occurred from 100 to 300 miles from Ames, and as word was not sent until one or more deaths had occurred, and then by mail, it is evident that with the utmost promptness from one to four days must elapse after the time of the first death until the examination was made. It is a significant fact that in 1892–93 only one outbreak occurred near Ames, and in this instance one of the two animals that died was found dead and the other was sick but two hours. However, several outbreaks were visited, and post-mortem examinations were made on a number of animals within a few hours after death.

The method of investigation followed in 1892 was to make careful post-mortem examinations, to make a large number of cultures from the different organs of all the animals that were in a suitable condition, to preserve in alcohol or Müller's fluid pieces of the tissue for microscopic examination, and to take to the laboratory at Ames large pieces of the organs for microscopic study while they were in fresh condition, and for additional cultures when deemed advisable. A large number of cover-glass preparations from the blood[1] and various organs were also made at the post-mortem, and subsequently stained and examined microscopically.

In 1893 Dr. F. L. Kilborne investigated several outbreaks of the disease in Iowa. He made the post-mortems, and sent cultures and tissues to the laboratory of the Bureau, where they were carefully examined. Although this method was attended with many disadvantages, it was in a measure quite satisfactory.

From the symptoms of this disease reported in 1892, the possibility of its being an affection of the nervous system resembling rabies to a certain extent was suggested. This theory was strengthened by the existence of a disease among cattle at Green, Iowa, in the fall of 1892, which could not be differentiated from rabies by the symptoms manifested or by rabbit inoculations, and in which a history of a dog bite could not be obtained. (See page 71.) The determination of this point by rabbit inoculations, the repetition of the bacteriological work, and the carrying out of certain feeding experiments for the purpose of testing the extent of the infection in the cornfields where the disease occurred were the lines of investigation followed in 1893.

The absence of recorded data on this disease has led to a somewhat detailed account of the conditions under which the various outbreaks occurred, and the appearances found upon post-mortem examination of certain of the animals. In the history of the outbreaks, as well as all facts relating to the individual animals prior to the post-mortem examinations, we must rely necessarily upon the statements of the owners who were carefully questioned concerning them.

OUTBREAK NO. 1.

This outbreak occurred on the farm of Mr. John Bixler, located about 3 miles west of Red Oak, Iowa. Mr. Bixler had about 70 animals (calves, cows, and steers) which had pastured on prairie land during the summer. About the 1st of November the cattle were turned into cornstalk and clover fields (20 acres of each). For the first six days they were allowed to remain daily in these fields one and one-half hours in the morning and the same time during the afternoon. After six days they were allowed to run in the cornstalk field, meadow, and pasture at will. It was observed, however, that they remained almost entirely in the cornstalk and clover fields. All the fields were on upland. The cattle had plenty of salt and well water.

November 11 the cattle were yarded for dehorning purposes, and each animal was noticed. They all appeared to be well. On the following morning a 2-year-old steer was found lying in a natural position, excepting the head which rested on the ground and moved continually from side to side. He could not be induced to get up. The frost on the grass showed that he had not floundered about. He remained in this position throughout the day and died early in the night.

---

[1] In addition to these, fresh preparations of blood were made and sealed, so that they could be examined microscopically before drying. It was found that white wax could be used with much success in sealing these mounts. Blood could be kept fresh for from eighteen to thirty hours by this plan.

November 14, the cattle were driven to water about 11 o'clock in the morning. A 2-year-old steer was observed to tarry behind the others. At 3 o'clock he was found lying in a dying condition. He was very much bloated, and died without struggling. No other cattle died, although they continued to run in the same fields. Notice of this outbreak was not received for three days after the last death had occurred. Owing to the fact that the animals had been skinned immediately after death, and that the temperature had been below freezing, the post-mortem changes were not so far advanced as would ordinarily be expected. They were examined November 18.

NO. 1 (STEER 2 YEARS OLD).

This animal had been dead five days and was much bloated. Brain appeared to be normal; no evidence of congestion. There was a slight pigmentation of the pia[1] over the frontal lobes. First and second stomachs were normal. Third stomach very hard; could be dented with the thumb only with difficulty; contents very dry; mucosa sloughed off (probably due to post-mortem changes). The mucosa of the true stomach deeply reddened in places, apparently due to blood extravasation. The mucosa of the duodenum and upper jejunum reddened, sprinkled with hemorrhagic areas, and covered with a muco-purulent substance. The edges of the folds of the mucosa in the cæcum and colon also reddened. Rectum appeared to be normal. Liver, spleen, and kidneys discolored and soft, due largely to decomposition. Lymphatics normal. No hepatization of the lungs.

NO. 2 (STEER 2 YEARS OLD).

This animal had been dead nearly four days. He was bled while in a dying condition, and consequently the post-mortem changes were less advanced than in No. 1. The brain was pale; small amount of pigment over frontal lobes. The third stomach was firm; contents dry. The fourth stomach and intestines showed more intense congestion than in No. 1; no hemorrhages. Gall bladder contained considerable thick, viscid, greenish-yellow bile. Kidneys pale. Spleen and lymphatics appeared to be normal. Liver reddened; edges rounded. Lungs not diseased.

Bacteriological examination.—Cultures were made from steer No. 2 only. Tubes of agar were inoculated with pieces of the brain, liver, and kidney. The tubes inoculated from the kidney remained clear. The others developed into pure cultures of *Bacillus coli communis.* A rabbit inoculated subcutaneously with 0.5 c. c. of a bouillon culture remained well. This organism was studied carefully, but found to possess no properties unusual for the colon bacillus.

Cover-glass preparations were made from the various organs at the autopsy. Those made from the kidney and liver contained putrefactive bacteria. In other preparations no bacteria were discovered.

Microscopic examination.—No microscopic examination was made of the organs from No. 1. The brain, liver, and kidneys in No. 2, examined in a fresh condition, showed advanced post-mortem changes. There was an abnormal amount of blood in the liver, cell nuclei visible, cellular protoplasm granular. Epithelium of the kidneys granular. Stained sections of the liver from pieces hardened in alcohol exhibited a granular condition of the cells; the nuclei were broken up but their outline was unchanged. There were few free nuclei. In areas involving a variable number of lobules the intralobular capillaries were distended with blood, the liver cells being separated from each other. The blood engorgement was more intense in the inner zones of the lobules. In similar sections of the kidney a few of the capillaries were distended with blood. Epithelium granular (probably due to post-mortem changes).

---

[1] This pigmentation is not uncommon in healthy cattle, as determined by the examination of the brains of a large number of animals slaughtered for beef.

In sections of both liver and kidney stained with aniline dyes a few putrefactive bacteria were observed.

OUTBREAK NO. 2.

Mr. E. E. Francis, residing near Wales, Iowa, lost one heifer in a cornstalk field. October 29 Mr. Francis turned 56 cattle in a cornstalk field for about two hours, when they were returned to the pasture. Thereafter the cattle were in the field continuously, and although they had access to a meadow they were observed to remain in the cornstalks most of the time. The corn was on upland. There was an unusually small amount of smut. The cattle had plenty of salt and well water. November 1, three days after the animals were turned into the cornstalk field, a heifer appeared to be dull, and the next morning she was found dead. She was examined by Mr. Francis, who found the rumen full and quite hard. No other lesions were noticed by him. The remaining cattle continued to run in the cornstalk field, but no other deaths occurred.

OUTBREAK NO. 3.

This outbreak occurred on a farm worked by Mr. L. W. Williams, located 3½ miles from Ames, Iowa. Mr. Williams had 13 acres of corn. It was on upland with the exception of a low, tile-drained area of about one-fourth acre which was said to have been wet during the spring and early summer. On this area the corn was small, but was said to have had a good color. The corn throughout the field was thin (few stalks in each hill) and the stalks large. There was very little smut.

November 22, Mr. Williams turned seven cattle, all in good condition, into the cornstalk field. The herd was composed of two cows, one 3-year-old and four 2-year-old heifers. The cattle were left in the field all day, but yarded at night without food. They had plenty of well water, but no salt until November 26. For the first two days they had access to a millet field, after which they had no food other than the cornstalks. Prior to turning the cattle into this field they had been in a clover meadow. The animals did well and no ill effect was observed until November 27, when a 2-year-old heifer was found dead.

November 29, the 3-year-old heifer was apparently well at 5.30 a. m. About 7 o'clock she was found resting squarely on her knees, the head extended and resting on the ground. She was frothing at the mouth and groaning as if in great distress. Mr. Williams said that blood oozed from the anus and vagina before death. She did not get up, dying about 9 a. m., two hours after she was first observed to be sick. The autopsies were made on both animals in the afternoon conjointly with Drs. Kilborne and Niles.

The remaining animals were kept out of the cornfield for a few days, after which they were returned without further loss. Mr. Williams had never before lost cattle with this disease, and so far as could be learned no other animals had died in that neighborhood while feeding in cornstalk fields.

Hogs which fed on the carcasses of the dead animals remained well.

No. 3 (A 2-YEAR-OLD HEIFER).

This animal was found dead November 27; examined in the afternoon of November 28. The post-mortem changes were considerably advanced. The spleen was slightly enlarged; a blood clot between the spleen and the rumen extended over an area 4 by 6 inches in diameter. The stomach and intestines throughout appeared to be normal. No impaction of the third stomach. Fibrous tufts on the ental surface of the omentum reddened from capillary injection. Liver discolored by post-mortem changes; no hemorrhages observable. About 100 c.c. of clear, viscid, reddish-colored bile in the gall bladder. A few punctiform hemorrhages beneath the capsule of the kidney; the blood vessels of the cortex slightly injected. The bladder was empty. Lymphatic glands contained a few small hemorrhages. Lungs normal. Punctiform hemorrhages beneath the epicardium over both ventricles. No blood extravasations detected beneath the endocardium. Several hemorrhagic areas along the aorta. The brain was normal in color and consistency.

Bacteriological examination.—A tube of agar and one of bouillon were inoculated with small pieces of the liver, and a tube of agar was inoculated from the kidney. The tube inoculated with the kidney remained clear. Those inoculated with the liver developed into pure cultures of a micrococcus. It possessed no pathogenic properties as determined by rabbit inoculations.

Cover-glass preparations were made from the liver, spleen, kidneys, and blood. Those made from the spleen showed, when properly stained, putrefactive bacteria. No bacteria could be detected in the others.

Microscopic examination.—The changes that had taken place in the tissues after death were so well marked that examinations were made of the kidney and liver only in the fresh condition. The parenchymatous tissue was granular, nuclei not distinct. In sections cut by the paraffin method the kidney exhibited no well-marked abnormal change. The nuclei of the tubular epithelium were quite distinct, but the cells were slightly swollen, and their free surfaces somewhat broken; the outline remained intact. The intralobular blood spaces were filled with blood.

No. 4 (A 3-YEAR OLD HEIFER).

The examination was made about four hours after death. The animal had been skinned, but the internal organs were not cold. Considerably bloated, said to be post-mortem.

The spleen was enlarged to fully twice its normal size. Hemorrhage between capsule and peritoneum extending over irregular areas, covering about one-third of the entire ental surface of the organ; pulp soft and of a uniform deep-red color; malpighian bodies were visible.

There was considerable light straw-colored œdema about the peritoneal attachments of the liver, the adjacent coils of the duodenum, and the gall bladder. The liver was much enlarged; edges rounded. The entire surface was mottled with purplish-black hemorrhagic areas varying from 1 to 10 mm. in diameter; frequently they were confluent. The intervening area was of a deep reddish color mottled with hemorrhagic infarcts varying from 1 to 6 mm. in diameter. Acini indistinct; parenchyma easily broken down. About 200 c. c. of a clear, not viscid, greenish-yellow bile. Beneath the mucosa of the gall bladder were several small areas of blood extravasation.

The kidneys were slightly enlarged; beneath the capsule there were a considerable number of punctiform hemorrhages. Capsule normal. Cortex thickened and the blood vessels injected; medullary portion apparently normal. Considerable hemorrhage between the kidney tissue proper and the connective tissue of the pelvis.

The contents and conditions of the mucosa of the rumen and recticulum normal. The contents of the omasum unusually soft. Mucosa normal. The mucosa over a few of the folds of the abomasum congested, otherwise normal. In the duodenum extending from the stomach for a distance of 3 to 4 feet the mucosa was sprinkled with punctiform hemorrhages, likewise similar areas in the jejunum. In the ileum and cæcum were several areas of congestion. Colon and rectum normal. Small blood clots in the uterus. Bladder empty. The mesenteric glands have hemorrhagic areas usually in the cortex, but in a few the medullary portion was involved. Lungs normal.

Heart of normal size. The blood vessels of the epicardium injected and blood extravasations along the course of the vessels which give the heart muscle a reddish (bloody) appearance. In the adventitia of the aorta were a large number of blood extravasations. These extended at irregular intervals from the heart to the diaphragm. The extravasations were more numerous over the auricles, beneath the endocardium, a large number of blood extravasations varying from mere points to areas a centimeter in diameter, many of them confluent. In the right ventricle extravasations covered about four-fifths of the entire surface. In the left ventricle a considerable area of its endocardium and auricle was hemorrhagic. Upon section the heart muscle of the right ventricle was found to be hemorrhagic for nearly one-third of its thickness, and in a few areas it extended entirely through the ventricular wall. A considerable number of blood extravasations in the muscles of the left ventricle. Several areas of the endocardium were of a grayish color. The blood in the heart and aorta was dark and firmly clotted.

Considerable hemorrhage was found over about four-fifths of the surface of the cerebellum and extending down between the cerebellum and cerebrum. The blood was between the pia and the brain substance. The blood vessels over the cerebrum were deeply injected; no hemorrhage. On section blood oozed from the cut ends of the smaller blood vessels.

Bacteriological examination.—Tubes of agar and those of either bouillon or glycerin-agar were inoculated, at the time of the examination, from the blood, brain, liver, spleen, kidney, and heart muscle. Large pieces of the various organs were taken to the laboratory in sterilized jars. From these, duplicate cultures were made on the following morning.

The cultures made from the liver, spleen, kidney, and one or more of those made from each of the other organs remained clear for several weeks, during which time they were shipped from Ames, Iowa, to Washington, D. C. Subsequently a few of them developed cultures of various species of saprophytic bacteria. The tubes of agar inoculated with the heart muscle, the blood, and brain developed into cultures of various bacteria within three days. The culture from the blood was impure. By means of agar plates two bacilli were isolated and subsequently carefully studied. They possessed no pathogenic properties and resembled in their cultural characters ordinarily observed saprophytic organisms. The agar tube inoculated with the heart muscle contained a pure culture of a nonpathogenic motile bacillus. The agar culture from the brain contained a streptococcus.

The agar culture from the kidney developed after several weeks into a pure culture of a motile bacillus. It was carefully studied, but found to be of no pathogenic importance as far as could be determined by inoculations into experimental animals.

A large number of cover-glass preparations were made from each of the organs and subsequently stained by various methods for the purpose of demonstrating bacteria, but with negative results. The subsequent examination of sections of the various tissues preserved in alcohol and stained for bacteria gave similar results.

Microscopic examination.—Sections and teased preparations of all of the organs of this animal were examined in a fresh condition. The nearness of the animal to the laboratory rendered this examination unusually favorable. The lesions discovered, however, were very slight. The liver cells were usually somewhat granular, and surrounded by a large number of red blood corpuscles, which filled the intralobular blood spaces. The nuclei were visible but broken up, giving to each the appearance of an area more or less thickly sprinkled with deeply stained points. Changes could not be detected in the interlobular tissue. The spleen, lymphatics, and brain did not show abnormal changes in the cellular elements. The blood was very carefully examined in fresh preparations. The corpuscles appeared to be normal. In the stained sections of the organs preserved in alcohol and Müller's fluid and cut by the paraffin method the engorgement and rupture of the blood vessels (capillaries) was more distinct. In the liver and heart muscle this was especially well marked. In the liver the blood engorgement was intense in the central zone of the lobule. The outlines of the nuclei were not altered. In certain limited areas free nuclei were observed.

The parenchyma of the kidneys appeared to be normal. In certain limited areas the capillaries were distended with blood.

In the heart muscle the capillaries were injected about the hemorrhagic areas. The normal markings of the heart muscle in the thin areas were not effaced, the lesions being confined to the capillaries.

OUTBREAK NO. 4.

This outbreak occurred on the farm of Mr. S. P. Miller, who resided about 5 miles northeast of Red Oak. Mr. Miller had 14 acres of corn, all on high land. The corn was good, but there was considerable smut.

Mr. Miller stated that on November 15 he turned 24 cattle, mostly calves 4 to 7 months old, into the cornstalk field. For several weeks prior to this they had been on clover pasture. For the first three days the cattle were allowed to remain in the cornstalks for about three hours daily. They received salt once a week and were supplied with plenty of fresh well water. They were allowed to run at will in a clover meadow adjoining the cornstalk field and appeared to do well for eight days when they began to die.

November 23, eight days after the cattle were turned into the cornstalk field a calf was found dead and much bloated, with froth about the mouth and nose.

November 24, a yearling was found dead. It had taken water twice and appeared perfectly well the day before.

November 26, a calf was found unable to get up. It appeared to be stiff and could move only with difficulty. It appeared to suffer very much; head down and eyes rolled up. It kicked considerably, and was shot in the afternoon to relieve its suffering.

November 29, a calf was found sick in the morning. It appeared to be "crazy," butted its head against boxes, and made desperate efforts to get out of the barn. It laid down in the forenoon and could not get up. A part of the time it remained quiet, but at times it kicked and bellowed as if in great distress. In the afternoon there was a normal passage of the bowels. It ate nothing. At night it was killed by a blow on the head to relieve its suffering.

After the death of the fourth animal the remaining cattle were taken out of the cornstalk field and turned into a similar field on the opposite side of the road, where they remained well. Several horses were left in the infected field, none of which took the disease. Word was received of this outbreak at Ames November 28. November 30 we made an examination of these animals, but owing to warm weather the post-mortem changes were too far advanced in all but the last to make examinations of any value except to determine that the lungs were not hepatized.

No. 5 (A Calf 7 months old).

It had been dead about fourteen hours and was considerably bloated, but in good condition. The post-mortem changes were slight. The capsule of the spleen was apparently slightly thickened; pulp soft. The capsules and size of the kidneys were normal; cortex reddened, due to injection of blood vessels. Considerable blood escaped from the small vessels on section. About 750 c. c. of cloudy urine in the bladder. Liver of normal size. Centers of the acini of a deep reddish color; periphery pale. Considerable blood escaped on section. Interlobular tissue apparently normal. About 100 c. c. of clear, dark, viscid bile in gall bladder. Contents and mucosa of the first, second, and third stomachs normal. The contents of the omasum dry, but not impacted. The blood vessels in the duodenum were injected in areas for a distance of about 4 feet. Mucosa of the ileum deeply reddened; contents normal. In the mucosa of the cæcum, colon, and rectum were areas in which the blood vessels were deeply injected; contents normal. Lymphatics normal. Heart and lungs normal. Brain not examined on account of injury produced in killing the animal.

Bacteriological examination.—Tubes of agar were inoculated with small pieces of the liver, spleen, and kidney, and with several loops of the heart blood. About 20 c. c. of the blood were taken from the heart by means of a sterile pipette and transferred to a sterile cotton-plugged tube. It remained sterile. The tubes of agar inoculated with the spleen developed into pure cultures of a motile bacillus. One of the tubes inoculated with the blood developed into a pure culture of a motile bacillus belonging to another species. These bacilli were carefully studied and found to produce no pathogenic effect on rabbits. The other tubes of media inoculated from this animal remained clear.

A large number of cover-glass preparations from the different organs and blood were made. They were carefully stained and examined, but no bacteria or other microorganisms were discovered. The same results were obtained from sections of the kidney and liver stained for bacteria.

Microscopic examination.—Before it was possible to reach the laboratory the tissues taken from this case were too much decomposed for examination. The sections of the kidney and liver from pieces of those organs preserved in alcohol and Müller's fluid were carefully stained and examined. The kidney showed a slight injection of the capillaries in circumscribed areas. The liver showed in certain areas extensive engorgement of the intralobular blood spaces. In these spaces were a few free nuclei, and the liver cells were separated from each other. No appreciable change of the interlobular connective tissue. The spleen appeared to be normal.

OUTBREAK NO. 5.

This outbreak occurred on the farm of Mr. C. Johnson, of Tyner. He had two fields of corn. One of them contained 10 acres of upland and 2 acres of rather low, moist ground, and the other contained 45 acres of upland and 15 acres of low, wet ground. The corn on all the upland was very good; that on the lowland was "snapped" early in the fall and fed to cattle in the feed yard, all of which remained well.

October 31, 1892, Mr. Johnson turned 22 cattle into the 12-acre cornstalk field. Prior to this the cattle had been on pasture land. Before turning them into this field they were all dehorned. On the morning of November 1 a steer was found dead. The cause of death in this animal was attributed to the dehorning the night before, as the hemorrhage was extensive and the weather severe. No other deaths occurred while the cattle remained in this field.

November 19, the cattle were turned from the 12-acre field, together with 10 horses, into the 60-acre cornstalk field.

November 20, a 3-year-old heifer and a yearling steer were found dead. Both of these animals appeared well the night before.

November 25, two cows and two steers were found dead. One of the steers appeared to be "crazy and blind" the night before. The ground about the dead animals showed that they had rubbed their heads to and fro on the ground and that one of them had struggled considerably. Two of these animals were examined by a butcher, who stated that "the blood was a little dark, otherwise there was no disease." Mr. Johnson opened the heart and found dark colored clots of blood.

November 25 to 27, the remaining cattle were kept out of the cornstalk field.

November 28, they were allowed in the cornstalk field for about three hours.

November 29, they were in the cornstalk field all day.

November 30, a 3-year-old heifer which appeared to be well early in the morning was found lying down about 7 o'clock and unable to get up. She bellowed and kicked a great deal. When quiet, the legs were extended from the body and the tongue protruded from the mouth. She died two hours later.

Notice of this outbreak was received at the experiment station at Ames, December 1. In company with Dr. W. B. Niles I made a post-mortem examination on the dead animal (last to die), and carefully inspected the cornfield in the afternoon of the following day. Nothing was found to arouse suspicion. Arrangements were made with Mr. Johnson by which a feeding experiment in the infected field with other cattle could be made, but unforeseen incidents prevented the carrying out of this plan.

Mr. Johnson kept his cows out of this field for some days, when they were returned without further loss.

No. 6 (A 3-YEAR-OLD HEIFER).

This animal was examined about thirty hours after death. The organs were badly decomposed. The normal markings were for the greater part entirely effaced. There was no evidence discernible of ante-mortem changes in any of the organs. The third stomach was rather soft; feces in the rectum of normal consistency. The lungs reddened; no hepatization. Brain normal in color, but quite soft.

There were 10 horses, all of which remained well, in the cornstalk field with the cattle. Hogs which fed upon the carcasses of the dead animals remained well. The facts that the "snapped" corn was fed to fattening cattle without producing any disturbance would indicate that the death of the animals was not due to feeding upon the few nubbins of corn that were inadvertently left in the field. It was observed by Mr. Johnson that the cattle fed largely on the cornstalks on the lowland.

A large bundle of the stalks, mostly leaves, was collected from the low ground and sent to the laboratory of the Bureau for a chemical analysis and for feeding rabbits. (See page 42.)

OUTBREAK NO. 6.

Mr. Robert Johnson, residing about 4 miles from Mechanicsville, lost two steers after turning them into a cornstalk field. A few others were sick, but recovered. The history of the outbreak, as given by Mr. Johnson, is as follows:

Thirty-seven cattle, a flock of sheep, and six horses were fed on cut cornstalks from about the 1st of October until the middle of November. At that time all of the animals were turned into a small (3-acre) cornstalk field. They had plenty of salt and well water. The stalks were soon devoured, after which the animals were fed on cut cornstalks and tame hay.

In about five days after the cattle were turned into the cornstalk field, but while they were still feeding upon the standing cornstalks, two steers were taken sick and died in four days.[1]

While sick the steers ate with difficulty, frothed at the mouth, appeared to have sore mouths and eyes, and lost flesh rapidly. They exhibited no "crazy" symptoms.

November 21 a carload of the cattle was shipped to Chicago. Upon inspection at the stock yards one of the animals was rejected on account of its being "gaunt," but was sold to other parties. Soon after this two yearlings and a cow that were purchased and turned in with the remaining cattle were taken sick. They manifested the same symptoms but to a less degree than those which had perished. When examined, December 3, they appeared to have fully recovered. The horses and sheep remained well.

---

[1] It is highly probable that these cattle died from some affection other than the cornstalk disease.

OUTBREAK NO. 7.

This outbreak occurred on a farm worked by Mr. Michael Cosgrove, located about 7 miles east of Toledo, Iowa. Word of this outbreak was not received until several days after the last animal had died, so that post-mortem examinations were not made. The following facts concerning the cattle were obtained from the owner:

The last of October, 21 cattle were turned into a cornstalk field. They remained well while in this field. November 18 they, together with 6 horses, were turned into a cornstalk field of 30 acres, mostly upland. They were in the field for only a few hours each day for the first five to eight days; the balance of the time they were on pasture land. They had plenty of salt, and well water was kept continually within their reach.

November 27 a cow was found down and unable to get up. She died during the following night. During the next forty-eight hours two other animals died, after an illness of about twelve hours each. Each of the affected animals appeared to be in much distress.

The horses remained well.

The crop of corn was good. There was considerable smut, all of which was picked and drawn out of the field when the corn was gathered. In 1891 four steers were said to have died of this disease while in the cornstalk field on the same ground. Mr. Cosgrove believed that the smut was the cause of the disease, and hence the precaution taken in 1892.

OUTBREAK NO. 8.

Mr. C. N. Heberlee, of Stewart, lost five cattle in cornstalk fields during the fall and early winter. The history of the outbreak, as given by Mr. Heberlee, is as follows:

About November 10, 1892, 25 cattle, mostly yearlings, were turned into a 15-acre cornstalk field. For the first few days they were in the cornstalks for only a few hours each day, but later they were left in them continuously.

November 25, the cattle were transferred to a 20-acre cornstalk field. Both of the fields were on upland. There was a moderate amount of smut on the corn. The cattle obtained water from a small stream which ran through an adjacent field (creek bottom). Rye had been sown in the cornfield, which gave a considerable amount of green food. The cattle had not been salted since they were turned into the cornstalks.

December 1, three steers were found dead. They were last seen November 29, when they appeared to be well. They were examined by Dr. Kilborne December 1, 1892. Owing to mild weather decomposition was well advanced, and little could be learned of the pathological changes. The contents of the third stomach were soft. The mucosa of the duodenum in each case contained areas of deep congestion. The lungs were not hepatized. All markings of the liver and kidneys were effaced.

December 2, another steer (No. 7) was found down and unable to get up. It groaned frequently and died about 10 a. m. Later in the day another steer (No. 8) was found dead. He had evidently been dead for sometime. Both of the animals were examined by Dr. Kilborne in the afternoon of December 2.

No. 7 (A Yearling Steer).

This animal had been dead but a few hours and was in good condition, not bloated. The spleen was not enlarged; pulp firm. On the ental surface there were about twenty deeply stained fibrous tufts, similar to those found on the omentum in cases of Texas fever. Malpighian bodies distinct. Liver swollen, deeply congested, giving it a dark-red color. On section the acini appeared with dark-red centers with lighter peripheries. Gall bladder contained about 100 c. c. of clear, viscid bile. The cortex of the kidneys was pale; medullary portion apparently normal. The mucosa of the bladder was sprinkled with punctiform hemorrhages. The bladder contained about 1,500 c. c. of clear urine, which was found to contain a small amount of albumin.

The first three stomachs were normal. The mucosa of the abomasum and areas in the duodenum and jejunum were congested. Ileum was filled with a pasty substance for about 8 feet from the lower end. The mucosa hemorrhagic. The membrane of the cæcum, colon, and rectum deeply congested. Contents rather dry. Lymphatics œdematous, not congested. Lungs normal. Numerous blood extravasations beneath the epicardium over both ventricles. A few hemorrhages beneath the endocardium in the right ventricle; in left ventricle a large hemorrhagic area. Heart empty. Small hemorrhagic points observed in the subcutaneous tissue, especially in the axilla and groins.

Bacteriological examination.—Cover-glass preparations were made from the blood, liver, kidney, and spleen. They showed no bacteria. Tubes of agar and bouillon were inoculated with pieces of the spleen and liver and remained clear.

Microscopic examination.—No examination was made of the fresh tissues. Pieces of the liver, spleen, kidney, and thigh muscles were preserved in Müller's fluid and alcohol. Sections of these organs cut by the paraffin method were carefully examined. Those of muscle and spleen showed no abnormal changes. A few of the capillaries in the cortex of the kidney were distended with blood. The tubular epithelium normal. The intralobular blood spaces in the liver were distended with blood. The engorgement was more general than in the previous cases. Liver cells separated and a few free nuclei. In many cells the nuclei presented small, deeply stained points separated by unstained portions. The outline of the nuclei not changed. There was considerable destruction of the liver cells in the central zone of some of the lobules.

No. 8 (A Yearling Steer).

This animal was bloated; post-mortem changes well advanced. Considerable straw-colored œdema in connective tissue surrounding gall bladder. Mucosa of the fourth stomach (abomasum) deeply congested. The mucosa of the intestines appeared to be normal. In other respects the lesions, so far as they could be determined, were not different from those in No. 7. On account of decomposition cultures were not made and none of the tissues preserved for microscopic examination.

OUTBREAK NO. 9.

Mr. John G. Jones, of Wales, lost three young animals in cornstalk fields in November. When visited the cattle had been dead for several days and consequently a post-mortem examination was not made. The data concerning the management of the cattle, as detailed by Mr. Jones, is as follows:

The first week in November, 40 head of cattle were turned into a 10-acre cornstalk field. They were allowed to remain in the cornstalks a few hours only each day for the first few days. They had access to a 20-acre clover meadow, and were yarded at night. They received salt twice a week and well water every night and morning. The corn in this and the subsequent field into which the cattle were turned was on high, rolling, prairie land. The growth of the corn was good. There was a small amount of smut. About November 16 the cattle were transferred to a second cornstalk field, which contained 20 acres, all on upland. While in this field they received no food other than cornstalks. They were yarded at night as before.

November 23, about 5 p. m., a steer was found to be sick. He stood apart from the others. About an hour later an effort was made to get him into a shed, when he fell down and could not be induced to get up. He remained quiet until about 8 p. m., when he died, three hours after he was first known to be sick.

November 24 the cattle were all apparently well when they were turned into the field at 9 a. m. At 2 p. m. a steer was found sick. It died in about two hours.

November 25 the cattle appeared to be well when yarded at night. The following morning (November 26) a yearling heifer was found unable to raise her head from the ground, apparently in a semicomatose condition. She remained in this condition until death occurred some hours later. None of the cattle bloated before death.

The remaining cattle were kept out of the cornstalks on November 27 and 28, but they were returned November 29. No death occurred subsequently.

Four years before (1888) Mr. Jones stated that he lost 4 or 5 animals in cornstalk fields, and about eight years prior to that he lost 8 animals from the same cause.

OUTBREAK NO. 10.

Mr. David G. Jones, who lived on a farm adjoining that of Mr. J. G. Jones (Outbreak No. 9), lost 2 animals in a cornstalk field. The absence of the owner rendered it impossible to obtain a complete history of this outbreak. As near as could be learned 15 cattle were turned into a cornstalk field about November 18. November 24 a 2-year-old steer died. November 30 a yearling steer died about 6 p. m. It was examined by Dr. Kilborne on the afternoon of December 1, about eighteen hours after death.

NO. 9 (A YEARLING STEER).

The spleen was dark colored, not enlarged, pulp soft. The surface of the liver was mottled with irregular branching, yellowish areas. The same condition was found upon section. The remainder of the liver was of a deep reddish color; acini indistinct; gall bladder empty. The cortex of the kidney was congested; no blood extravasation. About 100 c. c. of apparently normal urine in the bladder.

The mucosa of the first and second stomachs was unusually dark; contents normal. Third stomach contents rather dry, but not impacted. The mucosa of the true stomach deeply congested. The mucosa and contents of the duodenum and jejunum normal. In the lower 3 feet of the ileum the contents were blood stained and the mucosa was deeply reddened, due to the injection of the capillaries; contents normal. Colon and rectum normal. There were extensive blood extravasations beneath the epicardium of both ventricles and beneath the endocardium in the left ventricle of the heart. A small number of ecchymoses in the right ventricle.

Bacteriological examination.—Owing to an accident, cultures were not made from this animal. Cover-glass preparations from the blood, spleen, liver, and kidneys showed no bacteria.

OUTBREAK NO. 11.

This outbreak occurred on the farm of Mr. Tubbs, located a few miles from Emerson. It was learned at Emerson that the cattle had been dead for several days. This fact, together with the presence of a severe storm and deep snow, prevented a visit to the affected herd. The more general facts concerning the condition under which the outbreak occurred and the extent of the loss sustained were obtained from Mr. Tubbs, sr., who resided at Emerson.

About November 24, 140 cattle were turned into a cornstalk field of 65 acres. December 2, eight days later, 10 2-year-old steers were found dead. They were not known to have been sick. The remaining 130 animals were removed to a meadow, where 3 others died the following day. No more deaths occurred. The cattle were not returned to this field. The corn was good and situated on upland. There was very little smut.

About twenty-two years before Mr. Tubbs, sr., lost 20 animals in cornstalk fields. He had observed that the disease was most prevalent during dry seasons, and that snow or rain checked the progress of the disease. This opinion was entertained by veterinarians in that vicinity.

Mr. Tubbs reported that a neighbor, Mr. W. K. Post, lost 4 animals out of a herd of 33 which were turned into a cornstalk field. The deaths occurred on the eighth and ninth days after the cattle were turned into the field. Six young horses were pastured in the field with the cattle. The horses and the remaining cattle were kept continually in the cornstalk field, but no other deaths occurred.

OUTBREAK NO. 12.

This outbreak occurred on the farm of Mr. C. W. Hunt, of Woodbine. Although Mr. Hunt was visited by Dr. Kilborne immediately after receiving notice of the presence of the disease among his cattle, it was found upon arrival that no deaths had occurred for several days. Mr. Hunt's account of the symptoms observed in the affected animals, together with his statement concerning the condition of cornstalks and his method of managing the cattle, are unusually interesting.

During the first week of November, 33 cattle were turned into a cornstalk field of 20 acres. For the first three days the cattle were in the cornstalks daily for two, three, and four hours, respectively. Afterwards they were in the field continuously. In about a week 2 steers were found dead. The remaining animals were left in the field until December 7, when they were transferred to a second cornstalk field. December 11 the same cattle were turned into a third cornstalk field containing 20 acres.

December 12 a steer was taken sick about 2 p. m. He laid down but could not get up. He remained quiet and was alive at dark, but was found dead on the morning of December 13, together with another steer which appeared to be well the night before.

December 13 a steer was observed to stop very suddenly and begin to tremble; muscles twitched and jerked. It soon laid down, and died in about an hour.

The symptoms generally appeared to Mr. Hunt to resemble those produced in a dog by an overdose of strychnine.

The cornfields were on high ground and all the corn was good. In the first two fields there was considerable smut and in the third a very large quantity. The cattle were watered from a running stream that passed through the place.

Mr. Beebe, a neighbor of Mr. Hunt, stated that he had lost three cattle in a cornstalk field. They died in about one week after being turned into the cornstalks.

OUTBREAK NO. 13.[1]

This outbreak occurred near Van Meter, Iowa. Mr. A. B. McCleary purchased the standing cornstalks in two adjacent cornfields, containing 25 and 20 acres, respectively. They were on high ground. October 27, Mr. McCleary turned 35 cattle, mostly 2-year-old steers, into the 25-acre field. The cattle had free access to salt, which was kept continuously in an adjacent yard. They obtained drinking water from a small stream which passed along one side of these fields. The cattle were allowed to remain in the field continuously until November 26, when they were turned into the adjacent field. They also had free access to the first field, and obtained salt and water as before. The growth of the corn was much better in the second field than in the first. The soil was apparently identical, and the difference in the growth of the corn was attributed to the effect of the previous crops. In the first field the corn crop was preceded by oats and in the second field by clover meadow. There was a moderate amount of smut on the corn in both fields.

November 28, two days after the cattle were turned into the second field, a steer was found dead.

Early in the morning of November 29, 2 steers were found lying down and unable to rise. About 11 a. m., 2 others were found to be sick, and in a short time were unable to stand. One of the first affected was found dead at noon. The other 3 died before 2 p. m. Between 11 a. m. and 12 m. Mr. McCleary transferred the remaining 30 steers to the stock yard of his father, about 1 mile distant. About 1 p. m. the sixth steer was taken sick in the stock yard and died in about six hours. At 5 p. m. a seventh steer (No. 10 examined) was taken sick and died within two hours.

---

[1] The field notes on this and subsequent outbreaks were made by Dr. F. L. Kilborne in the fall of 1893.

November 30 a steer (No. 11 examined) was found dead at 5 p. m., and it had evidently been dead but a short time. It was apparently as well as usual at 11 o'clock the night before.

Five colts, 2 and 3 years old, were in the cornstalk field with the cattle, but they remained well and were left in the field for three or four weeks after the cattle were removed.

The symptoms were closely observed in this outbreak by Mr. McCleary, his father, and two brothers, and also by a local veterinary practitioner.

The affected animals were quiet and stood with the head down. If forced to move they walked with jerky movements, especially of the hind limbs. In one case a steer showed a marked desire to press his head against the side of the stable, where he stood until he fell and was unable to rise.

Soon after the attack the animals showed marked weakness, especially in the knees, causing them to stagger if they tried to walk. After a variable time they would fall on their knees, shake the head, and either rise with some difficulty or fall completely down. Often two or three efforts to rise would be required before succeeding.

Following the first symptoms of weakness, they shivered or trembled "as if suffering from a chill." They were not feverish to the touch. Dribbling of the urine occurred while the animals were staggering about just before falling.

After falling down and getting up a few times they would fall on one side, unable to rise, and stretch out at full length, with the head thrown backward. Respiration was accompanied by "groans," as if the animals were in great distress. More or less saliva escaped from the mouth after the animals fell. The limbs and head, but especially the former, moved continuously backward and forward. No excitement or other nervous symptoms were noticed in this herd. The animals began to bloat soon after death.

The most reliable account of a recovery which we have from an attack of the cornstalk disease occurred in this outbreak. A steer was taken sick about 2 p. m., November 29, with the same symptoms as those manifested by the animals which died. He had difficulty in walking and trembled violently for a short time, when he fell. After ten or fifteen minutes he succeeded in getting up. He staggered about for a short time, when he began to improve and made a rapid recovery.

November 30, post-mortem examinations were made on two of these animals.

No. 10 (A 2-YEAR-OLD STEER).

This animal had been dead for about twelve hours when examined. Post-mortem changes were well marked. The rumen was distended with gas. Spleen considerably engorged, the pulp soft, and the normal markings were barely visible. There was considerable blood extravasation beneath the capsule.

Liver congested; surface, especially around the border, mottled with deep-red points. The acini, especially as seen on section, were distinct; the central portion exhibited a dark-red color; the peripheral portion was quite pale. Bile of a dark-greenish color and normal consistency. Kidneys apparently normal; urine cloudy. The first three stomachs normal in appearance. In the true stomach the mucosa was hyperæmic and sprinkled with numerous petechiæ. Intestines apparently normal. Lungs normal. Numerous points of blood extravasation beneath the epicardium. A few ecchymoses beneath endocardium of both ventricles. Both ventricles were filled with blood clots. Brain somewhat softened, otherwise normal.

Bacteriological examination.—Tubes of agar and glycerin agar were inoculated with pieces of the spleen, liver, kidney, brain, and several loops of the blood. The tubes of glycerin agar inoculated with the spleen and blood, and the agar tube inoculated with the liver developed into pure cultures of a micrococcus. The other tubes that were inoculated from this animal remained clear. The micrococcus was found to possess no pathogenic properties as indicated by intravenous inoculation of a large quantity of a pure culture into a rabbit.

Cover-glass preparations were made from the different organs and tissues at the autopsy. They did not reveal the presence of any bacteria. The same is true of sections of the organs preserved in alcohol.

The brain was placed in pure glycerin and shipped by express to the laboratory, where it was received three days later. Several rabbits were inoculated beneath the dura with from one to three drops of a suspension of the brain substance in sterilized distilled water. The inoculation wound healed rapidly and the rabbits remained well.

No. 11 (A 2-YEAR-OLD STEER).

This animal had been dead about thirty hours and had already been opened by Mr. McCleary and the intestines and stomach removed. The decomposition was somewhat advanced. The liver was deeply congested and presented acini with dark centers and pale periphery, as in No. 10. The capsule of the spleen was dotted with several pyriform hemorrhages. Pulp very soft and almost black (probably post-mortem). Brain substance of normal consistency, a considerable quantity of semiclotted blood surrounding the cerebellum beneath pia. A few punctiform hemorrhages in meninges covering dorsal surface of the spinal cord in the cervical region.

OUTBREAK NO. 14.

Mr. A. N. Hadley, of New Providence, lost 15 animals in cornstalk fields. He had lived on the farm for seven years, but this was the first appearance of the disease. Unfortunately word of this outbreak was not received until several days after the last death occurred. The history of the outbreak, as learned from Mr. Hadley, is as follows:

October 25, 38 cattle, mostly 2-year-old steers, were turned into a 20-acre cornstalk field and allowed to remain there continuously. October 30, 7 of the steers were found dead and at 2 p. m. 3 more had died. On the following morning (October 31) another steer was found dead. The remaining cattle were turned from this field into a cattle yard and fed hay together with a little corn on the ear. After three or four days' stay in the yard they were turned into a second cornstalk field containing from 25 to 30 acres. At first they were kept in the field for from two to three hours daily, but after three days they were allowed to remain in the cornstalks during the day and were yarded at night.

November 15, the cattle were turned into a third field containing 20 acres, adjacent to the second one. As in the second field, they were allowed to remain in the cornstalks for only a few hours each day for the first few days. November 22, a steer was found dead in the morning and a second one died in the evening. The animals were known to have been sick for three or four hours. From November 23 to November 29, the remaining cattle were kept in a yard and fed as before. November 29, they were turned into field No. 4, which contained 35 acres, and were allowed to remain in this field continuously until December 7, without further loss.

December 2, 14 yearling steers were purchased and turned into the fourth field with the other cattle. December 7, all of the animals were turned back into field No. 3. There was from 6 to 8 inches of snow on the ground. December 9, two days after the cattle were turned into this field, two of the recently purchased yearlings were found lying on the ground unable to rise and died in a few hours. The death of these animals occurred in seven days after they were turned into field No. 4. After the death of these two animals the cattle were removed from the cornstalk fields.

The cornfields were on upland, excepting a narrow strip above a water course which passed through each field. In the first, third, and fourth fields the corn was good and the growth of stalks large, but in the second one the growth of the stalks was much smaller.

While in the first field the cattle had also the run of several acres of grass land from which a crop of hay had been removed. The first field was about a mile distant from the other three, which were adjacent to each other. There was only a small amount of smut in the first, second, and fourth fields, but in the third there was a much larger quantity. The cattle were salted irregularly while in the first field. Subsequently salt was constantly in the fields. While in the first field the cattle were supplied with water from a small stream which passed through it; in the other three fields they obtained water from a tank in an adjoining yard. Much of the corn from the first field had been "snapped" and was being fed to fattening cattle at the same time that the 35 animals were pasturing in the cornstalks. The "snapped" corn produced no ill effects.

A few symptoms were observed by Mr. Hadley. One of the animals (eighth to die) appeared to have a chill which lasted two hours before death. This animal, however, with several others, had broken into a neighbor's cornfield the day before, and the cause of death was evidently complicated with, if not due to, overengorgement with corn. This may explain the cause of the bloating.

OUTBREAK NO. 15.

This outbreak occurred on the farm of Mr. C. Keyser, of Percival. He had 80 acres of corn on "bottom" land, which had been under cultivation for many years and the stalk field was free from weeds. The corn was good. There was a moderate amount of smut.

November 26, 100 cattle, consisting of young cows (2 to 5 years old) and a few steers (2 to 3 years old), were turned into the cornstalk field and left there continuously. For a month prior to this they had been in another cornstalk field. December 2, 10 young cows were found dead. The remaining cattle were taken from the field, but after a day or two were returned. Twelve of these died during the next four days. The cattle were being watched when 3 or 4 of the cows were taken sick. They were reported either to move about strangely or stagger more or less for a few minutes, then fall down and die within an hour or two. December 4, 75 of the cattle were sold and shipped to Omaha.

Although the infected farm was visited as soon as possible after word of the appearance of the disease was received, the last animal to die had been dead for three days, and on account of advanced decomposition post-mortem examinations were not made.

OUTBREAK NO. 16.

This outbreak occurred on the farm of the French Community, near Corning. November 20, 75 cattle, consisting of 33 yearlings, 7 2-year-olds, and 35 cows, were turned into a cornstalk field for from ten to fourteen days. The yearlings remained in the field continuously, but the cows were yarded every night.

November 26, a 2-year-old heifer died. The other animals appeared to be well.

November 29, 7 yearlings were found dead. One yearling and a 2-year-old were sick. The yearling died in a few hours, but the 2-year-old recovered. None of the cows were affected.

About 60 acres of the corn were on upland and about 20 acres were on lowland. The cornfield on the upland and the one on the flats were separated by a considerable area of blue-grass pasture land, which was for the greater part on the flats. The corn was good. That on the lowland contained considerable smut, but there was very little on the upland. This was the fifth consecutive crop of corn on this land. The cattle had free access to a tank of water standing in the yard and supplied from a well by means of a windmill. The cattle were regularly salted every two or three days.

Mr. L. Brettanier, the herdsman for the community, observed the following symptoms in the first heifer to die: When first observed she was standing apart from the others "acting strangely;" her eyes had a wild or startled look, and at the same time she acted a little "crazy." In walking she would raise her hind feet with a peculiar jerky movement. She soon began to stagger, and after a few minutes laid down and was unable to rise. She continued to move her head and limbs back and forth until death ensued.

OUTBREAK NO. 17.

This outbreak occurred on the farm of Mr. John Sunderland, of Hastings. He had 70 acres of corn, all on upland. The corn yielded a good crop and there was a heavy growth of stalks. It was the second year that corn had been cultivated on this land. There was considerable smut this year, but in 1892 there was very little.

December 3, 70 head of cattle (35 yearlings and 2-year-olds and 35 from 3 to 5 years old) and 10 horses were turned into the cornstalk field.

December 7, 7 of the young cattle were found dead. No deaths occurred subsequently. The horses remained well.

In 1892, 15 of a considerable number of animals pasturing in the cornstalks on this same land died. As in 1893, they were all young cattle, the older animals being apparently exempt from the disease.

OUTBREAK NO. 18.

This outbreak occurred on the farm of Mr. E. M. Carey, of Red Oak. He had 40 acres of corn on river-bottom land. There was a heavy growth of stalks and the corn was good. Previous to this year clover and timothy had been raised on this ground. When the field was examined (December 17) very little smut could be found.

December 7, Mr. Carey turned 226 cattle (about half of which were yearlings) into the 40-acre cornstalk field where they were allowed to run in the field continuously.

December 10, a 2-year-old heifer died very suddenly.

December 14, 16 yearlings and one 2-year-old were found dead. All of these animals were apparently well when they were seen on the preceding morning by the foreman of the farm. No deaths occurred subsequently, although the cattle were left in this field for several days. The dead yearlings were about equally divided between the two sexes, and one of the other cattle was affected. The cattle obtained water from the river, and were not salted. The cornfield was free from grass, and the animals had no food other than the corn fodder.

December 17, an examination was made of the dead animals. The post-mortem changes were too far advanced in most of the cases to enable the identification of slight pathological changes. In one case, however, the organs were quite well preserved.

NO. 12 (A YEARLING STEER).

This animal died on the morning of December 14, and was examined early in the morning of December 17. Post-mortem changes not as advanced as would naturally be expected. The liver was congested. The acini were distinct; the central portion was of a bright reddish color. The bile was normal in appearance and quantity.

The spleen was not enlarged; there were several ecchymoses beneath the capsule. The kidneys and urine appeared to be normal. The intestinal tract appeared to be normal excepting the omasum, which was unusually firm, although not impacted. The lungs were normal. There were several petechiæ beneath the epicardium, and post-mortem clots in both ventricles.

The brain was considerably softened, otherwise apparently normal. In one other case the liver, spleen, and heart exhibited lesions similar to those in No. 12. Intestinal tract normal.

Bacteriological examination.—Cover-glass preparations were made from the different organs and subsequently examined for bacteria. They gave negative results. Tubes of agar were inoculated with pieces of the liver, spleen, kidney, heart blood, and brain. December 21, the inoculated tubes were all clear. December 28, one of the agar tubes inoculated from the blood had developed into a pure culture of a spore-bearing bacillus belonging to the *Bacillus subtilis* group of bacteria. All of the other inoculated tubes remained clear.

About 3 c. c. of the blood were drawn up into a sterilized pipette and sealed. A rabbit inoculated subcutaneously with about 2 c. c. of this blood remained well. Four rabbits inoculated beneath the dura with from one to three drops of a suspension of the brain in bouillon remained well.

CORNSTALKS AFFECTED WITH "CORN BLIGHT," OR THE BURRILL DISEASE ARE FREE FROM PATHOGENIC PROPERTIES.

The large number of bacteriological examinations of the organs of cattle which died in cornstalk fields in the fall of 1892 showed conclusively that there was no single species of bacteria constantly present in the organs of these animals that could be cultivated under ordinary conditions. The evidence was convincing that if the bacillus of the corn disease did invade the organs of cattle it was either destroyed or rendered incapable of subsequent development on media satisfactory for the growth of the bacillus isolated by Professor Burrill. From Billings's report it is evident that the bacillus which he described is cultivated without difficulty. Billings, however, traced the origin of this disease in cattle to the affection of cornstalks described by Burrill[1] in 1889. The theory which he advanced was that the bacillus in the affected corn ingested by the cattle produced rapidly fatal results (septicæmia).

A review of Professor Burrill's description of the disease in the cornstalks shows that it attacks (1) the young corn in which the roots are first affected, and (2) the full-grown stalks after midsummer, when it manifests itself by certain discolored areas, more particularly on the leaf sheaths. In the young plant the disease, which is restricted to certain limited areas, dwarfs the stalks, resulting in the loss of the crop, while in the mature plant it is widely distributed and does not appear to be possessed of economic importance. The same species of a bacillus was isolated by Burrill from both the diseased root of the young corn and the lesions on the leaf sheath of the mature stalk. Billings[2] also states that "this corn disease is exceedingly local in its extension, being not only limited to certain fields, but even to certain portions of such fields and most probably to certain stalks in a hill, or at least to individual groups of stalks." It appears, therefore, that Billings recognized but one form of the corn disease and his illustrations[1] show that to be the disease in the mature plant. It appears, therefore, that he mistook the disease in the full-grown corn, which is exceedingly common, for the more rare affection manifested in the dwarfed stalks, which he considered responsible for the death of the cattle.

---

[1] Those interested in the disease of corn are referred to Professor Burrill's paper on a New Bacterial Disease of Corn, Bulletin No. 6, University of Illinois, Agricultural Experiment Station, 1889; Proceedings of the Society for the Promotion of Agricultural Science, 1889, 19.

[2] Loc. cit.

This reported scarcity of the disease of corn said to be fatal to cattle, the failure to find a specific organism in the organs of the cattle examined in 1892, and the rapidity of the disease with symptoms said to resemble those of poisoning, led to the supposition that possibly animals which died while feeding in cornstalk fields might be poisoned from the bacterial products stored up in the diseased cornstalks. This led to an investigation of the cornstalk disease in its relation to the affection of cattle.

In August, 1893, I consulted Professor Burrill at Champaign, Ill., in reference to the corn disease which he had described. He very kindly offered me the use of his laboratory and personally pointed out the disease which he had described in the full-grown corn, and indicated the character of the lesions from which he predicted I would obtain pure cultures of the bacillus which he had found. It was too late in the season to study bacteriologically the dwarfed corn (result of the disease in the root of the young plant), and consequently my investigation of that form of the disease was limited to certain feeding experiments hereafter described. The examination of the disease in a large number of cornfields showed the lesions to correspond with the description of the disease in the mature plant as given by Professor Burrill, and consequently a further account of its appearance is not necessary.

A large number of agar and glycerin-agar cultures[2] were made from the lesions indicating the first stages of the disease in the leaf sheaths from cornstalks taken from fields at or near Champaign, and also from similarly diseased corn from several other localities, more especially about Kankakee and Mazon. A few cultures were made from the dead roots of the dwarfed corn and from the erosions (old lesions) on the leaf sheaths, but these invariably developed, as predicted by Burrill, impure cultures often containing fungi which, on account of their rapid growth, caused the cultures to be abandoned.

---

[1] The technique employed in making the cultures was very simple. The cornstalks were selected which showed from the exterior minute discolorations on the leaf sheaths. These were cut close to the ground and taken to the place where the cultures were made. The leaves and upper part of the stalks were then removed. The lower part was washed in a one-thousandth solution of corrosive sublimate. The closely fitting leaf sheaths were then removed by making first a deep circular incision just above the node, from which the diseased sheath was removed entirely or in part by means of gentle pressure with sterile forceps. With sterile (flamed) instruments a bit of the affected parenchymatous tissue was removed and placed in the culture media. It is of interest to add that a platinum loop rubbed over the inner surface of the freshly removed but tightly fitting leaf sheaths and then drawn over the inclined surface of agar in most cases showed these surfaces to be germ free. When the sheath was loose, however, its inner surface became contaminated with bacteria and fungi.

[2] Bulletins 22 and 23, Nebraska State Experiment Station.

A careful examination of a large number of cornfields situated at short intervals across the central and northern parts of the State of Illinois showed the disease in the mature corn to be exceedingly abundant. Later observations revealed its presence in the States of Indiana and Maryland, and it is highly probable that it is even more widely distributed.

The dwarfed corn was found to be quite rare. In addition to the statement that this was limited to certain circumscribed areas, it was learned that these areas always produced dwarfed cornstalks when the crop was repeated year after year. Mr. McClure, assistant horticulturist at the State University of Illinois, directed my attention to this disease on the farm of Mr. Hay, near Kankakee, Ill. I found in his cornfield a large number of areas, varying in size from a few square rods to an acre or more, upon which the corn was dwarfed and worthless. This was considered by both Mr. McClure and Professor Burrill to be a most typical example of the effect of the disease attacking the root of the young plant.[1]

About 80 per cent of the cultures made from the first stages of the disease in the leaf sheaths contained a bacillus not distinguishable from the one described by Burrill. From the other cultures four species of saprophytic bacteria were obtained frequently in pure cultures. Although the bacillus described by Burrill was found in large numbers in the diseased cornstalks, its absence from the organs of the cattle dying in the cornstalk fields in 1892 rendered it desirable to test the pathogenesis of the affected cornstalks themselves. It was the intention to select thoroughly infected cornstalks and to feed them to cattle without an admixture of other food. The necessity for this artificial experiment, however, was eliminated by existing conditions brought about by more natural causes.

A drought which extended over the greater part of the State of Illinois in August and September, 1893, dried up the pastures and compelled the farmers to feed their cattle. In many instances cornstalks were the only food given. As all of the corn was found to be affected with this disease, it was evident that no feeding experiment could be planned that would be more efficacious than those already being made. Several herds of cattle that were fed exclusively on these stalks were carefully watched for a period of several weeks, but no evidence of disease appeared. The cornstalks fed were carefully examined and found to be thoroughly infected with the Burrill disease. It should be noted that the cattle ate these stalks much closer than they did dried ones in the field later in the fall, and consequently they ingested many more of the diseased areas. It is of interest to add that so far as it was possible to learn, the cornstalk disease did not appear among cattle in 1893 in the districts visited in the fall and where the mature cornstalks were known to be badly affected by the Burrill disease. Unfortunately, no feeding experiment with the dwarfed corn was carried out with cattle. An experiment was planned for late in the fall with the dwarfed stalks from Mr. Hay's field, but a severe storm destroyed the stalks to such an extent that it was impossible to complete it. Enough of the blighted stalks were collected, however, for a rabbit experiment.

---

[1] I was first informed by Mr. McClure, and subsequently by others, that the germ theory of the dwarfing of the corn was losing ground and the previously entertained one, that it was due either to animal parasites or to a lack of "something" in the soil, was being revived. A careful study of this form of the disease is being made by both Mr. McClure, of Champaign, and Mr. Hay, of Kankakee. Mr. Hay is testing the efficiency of various fertilizers. He has found that a considerable portion of his land invariably yields dwarfed and worthless corn, while the land immediately adjoining it always produces a good crop. Thus far his experiments show that with a proper amount of fertilizers his land, which otherwise gave no corn, will yield a profitable crop. His experiments promise a practical method for preventing the loss now sustained from this cause.

FEEDING DISEASED CORNSTALKS TO RABBITS.

Dr. Billings[1] fed the leaves of the diseased corn to a rabbit with fatal results. From the organs he obtained cultures of a bacillus identical with the one which he had isolated from the diseased organs of cattle dying of the cornstalk disease. In his writings concerning the distribution of the bacteria in the stalks and the difference in the pathogenesis of the organism isolated from different portions of the plant we find the following remarkable statement:

> That it is in the stalks I myself have demonstrated by microscopic examination and cultures; but with this singular result: It is not disease producing when inoculated from this source in either rabbits or mice, while from the leaves it is, as we have seen from the feeding experiment in the black rabbit.

In the fall of 1892, a bundle of corn leaves and tassels was collected from an infected field (Outbreak No. 5) and sent to the laboratory at Washington for feeding rabbits.

Two rabbits were fed exclusively upon these leaves for a period of four weeks, during which time they remained apparently well. The animals had plenty of fresh water but no other food during this time. About three months after the feeding of the stalks one of the rabbits died. It was emaciated, but bacteria could not be found in cover-glass preparations from the organs, and culture media inoculated from them remained sterile.

In the fall of 1893, two feeding experiments were made with stalks known to be affected with the Burrill corn disease.

---

[1] Bulletin Nos. 7, 8, 9, and 10 (one volume), of the Agricultural Experiment Station of Nebraska, 1889, p. 134.

(1) Feeding of the leaf sheaths from the mature cornstalks.

(2) Feeding of the dwarfed stalks (disease in young corn).

In the early part of September several bushels of the badly diseased leaf sheaths were collected in the State of Illinois. Care was taken to select only those sheaths which were diseased and which presumably contained a very large number of bacteria. These were sent to the laboratory of the Bureau and used for feeding rabbits and for a chemical analysis. Likewise in the latter part of September a considerable quantity of the dwarfed yellow stalks, including leaves and roots, were received from Mr. Leon Hay, of Kankakee, Ill., whose cornfields contained many areas on which the corn was dwarfed and yellowish. Four rabbits were fed on each variety of the diseased cornstalks. The details of these experiments are given in the subjoined tables:

A.—*Feeding rabbits with the diseased leaf sheaths.*

Rabbit No.	Date feeding was begun.	Stopped feeding.	Remarks.
69	Sept. 6, 1893	Oct. 4, 1893	Emaciated; otherwise well.
70	do		Died Oct. 21. Emaciated; no other lesions observed; no bacteria discovered in the organs.
71	do	Oct. 4, 1893	Somewhat emaciated; otherwise well.
72	do	do	Do.

B.—*Feeding rabbits with dwarfed cornstalks.*

Rabbit No.	Date feeding was begun.	Stopped feeding.	Remarks.
73	Sept. 22, 1893		Died Oct. 17. Very poor; no lesions; cultures made from blood, spleen, liver and kidneys remain clear.
74	do	Nov. 1, 1893	Somewhat emaciated; otherwise well.
60	do	do	Do.
61	do	do	Do.

The rabbits were given plenty of water and all of the stalks (cut up) they would eat. They did well on the dwarfed cornstalks, but those fed only upon the leaf sheaths began to lose flesh after about two weeks, when they were given a small quantity of other food (wheat bran and oats) with the cut leaf sheaths.

The cause of death in rabbits Nos. 70 and 73 was not determined. It is highly probable, however, that it was due to malnutrition. The only abnormal condition that was discovered was marked emaciation. Tubes of agar and bouillon inoculated with bits of the organs and blood remained clear.

EXPERIMENTS TO ASCERTAIN THE EXTENT TO WHICH CORNSTALK FIELDS IN WHICH CATTLE HAVE DIED ARE DANGEROUS.

It was shown in the notes on the outbreaks of the disease in cattle that all of the animals that died in any particular cornstalk field did so in a very few days, and that the remaining animals were not appreciably affected. The cause of this peculiar condition can not be satisfactorily explained until the etiological factor is determined. A high degree of susceptibility of the animals to the normal constituents of the cornstalks would presumably give rise to such a phenomenon. If the cause is a specific one, such as a microorganism, the clinical history would indicate that the destructive agent was either concentrated, so that the cattle which perished were the only ones that had become infected, or that it was more generally distributed and that a tolerance was established in those which survived presumably by reason of small initial doses. An important and interesting fact is that where the number of cattle was small and the acreage of corn large, all of the deaths occurred before the cattle had fed to any extent upon the cornstalks. In view of these facts a few experiments were planned by which it was hoped to demonstrate whether or not the destructive agent was eliminated from the fields with the first deaths. The plan suggested was to turn cattle which had been either on pasture land or in cornstalk fields in which no deaths had occurred into the infected field immediately after the deaths in the first herd had ceased. As it was the general opinion among the farmers consulted that after the first deaths no further loss would be sustained, some difficulty was experienced in obtaining suitable cornstalk fields for this purpose and also in securing satisfactory cattle. In the fall of 1892 only one such experiment was made, and it proved to be of little value, as the owner of the cattle insisted upon removing them from the field on account of the severe cold weather and a heavy fall of snow. In the fall of 1893 the following experiment was carried out by Dr. F. L. Kilborne:

In Outbreak No. 13 (page 33), which occurred on the farm of Mr. McCleary, of Van Meter, 5 animals from a herd numbering 35 died while feeding in a 20-acre cornstalk field within four days after they were turned into it. The cattle were in the field five days.

December 5, five days after the cattle were removed from the field, 20 2-year-old steers were turned into it. Ten of these were from a herd that had been in cornstalk fields, located about a mile distant, for about five weeks. The other 10 were from the herd first turned into the field and had been yarded since November 29. These were returned to further test their power of resistance.

At the time the experimental cattle were turned into the field there was about 8 inches of snow on the ground, which did not entirely disappear until the close of the experiment. There was no snow on the ground at the time the cattle in the first herd died, but other outbreaks have occurred in the presence of a greater or less quantity of snow, so that presumably this condition had no influence on the result.

During the experiment the cattle obtained water and salt the same as those which suffered from the disease.

Before the experiment was begun a wagonload of stalks on which smut had grown was cut for a more specific feeding experiment with that article. Only about one-fourth of the husks, leaves, or tassels usually eaten by the cattle had been consumed by the first herd.

The 20 steers remained in the cornfield continuously until December 16, a period of eleven days, without the appearance of the disease. They were then removed.

THE BACILLUS OF THE BURRILL DISEASE OF CORN.

The observations and experiments with the diseased cornstalks already recorded are enough to show the innocuous nature of its supposed specific organism. There are, however, several interesting facts in connection with this bacillus. As previously stated, a bacillus was obtained in pure cultures from about 80 per cent of the media inoculated with the beginning lesions in the cornstalk disease. In cover-glass preparations they usually appeared in considerable numbers, especially in those made from the gum-like substance frequently found in the inside of the leaf sheath beneath the lesions.

Although Professor Burrill very satisfactorily described the bacillus he found, according to the requirements of that time, the development of new methods for differentiating bacteria shows that a positive identification of bacteria recently isolated with those previously described is a matter of much difficulty, excepting where definite and characteristic pathogenic or physiological properties exist. In this case, however, I was fortunate in receiving from Dr. Smith a culture of a bacillus originally obtained from a culture of the bacillus of the cornstalk disease sent to him by Professor Burrill. A comparative study of the two cultures enabled me to identify the bacillus which I had isolated with the one previously received from Professor Burrill. It was found, however, that these bacteria differed from the original description in their growth upon gelatin. They both liquefied this medium. The liquefaction took place very slowly, however, which may explain the statement in Burrill's description that it does not occur, as he failed to state the age of the culture when last examined. As these bacteria in other respects correspond with the original description, this variation under the existing conditions does not appear sufficient for differential purposes. A study of these and the closely allied bacteria has shown that the power to liquefy gelatin and the quantity of gas produced by the fermentation of certain sugars varies somewhat as a result of continued artificial cultivation. The large amount of the disease in corn after midsummer led to a bacteriological examination of the soil from fields in Maryland, which resulted in finding very abundantly a bacillus closely related to the one obtained from the diseased cornstalks, and probably identical with it.

In 1893 Dr. Theobald Smith[1] identified the bacillus received from Professor Burrill and the one with which I compared my cultures as *Bacillus cloacæ.*[2] He also reports this organism from well water, both "polluted and unpolluted."

---

[1] Wilder Quarter-Century Book, 1893, p. 214.

[2] This bacillus was isolated from sewage and was originally described by E. O. Jordan. Experimental investigations by the State board of health of Massachusetts upon the purification of sewage, etc. Part II, 1890, p. 836.

The cultures of this organism obtained from different sources were carefully studied in parallel cultures and found to differ very slightly. The greatest difference between them was in certain physiological manifestations. As this bacillus has already been described it seems necessary at this time to point out only the more essential differential properties of the bacillus obtained from the diseased cornstalks.

DESCRIPTION OF THE BACILLUS.

Morphology.—A motile bacillus varying in length from 1.3 to 2 μ, ends rounded Appears in cultures singly or united in short chains or clumps. From five to fourteen flagella have been demonstrated. It stains readily with aniline dyes.

Cultural characters.—It develops a grayish, somewhat vigorous, glistening, nonviscid growth on the surface of agar. In the depth of agar it is more feeble. Gelatin is softened along the needle track and beneath the quite vigorous grayish growth which appears on the surface at the end of six days. It liquefies very slowly. The liquid gelatin is clear, with a viscid, grayish sediment. Strongly alkaline in reaction. On potato a dull grayish, nonviscid growth appears within twenty-four hours. In alkaline bouillon the multiplication is quite active, the liquid becoming heavily clouded in twenty-four hours. The casein of milk is coagulated in about eighteen days. It possesses active fermentation properties. In the fermentation tube filled with bouillon containing 2 per cent dextrose the closed bulb is filled with gas in forty-eight hours. The liquid is very acid in reaction. The gas consists of 72 per cent CO_2 and 28 per cent H. In a similar tube containing saccharin the fermentation is quite as active, but the proportion of the gas constituents is different, being 66 per cent CO_2 and 34 per cent H. In bouillon containing lactose the closed bulb of the tube is not filled with gas until the fourth day. The gas is practically the same as that produced in the saccharin bouillon.

The pathogenesis of the bacillus isolated from the lesions on the cornstalks was tested by a large number of inoculations with pure cultures into rabbits, guinea pigs, and mice. The animals were inoculated by different methods and with varying quantities of culture. One rabbit which received 0.75 c. c. of a bouillon culture in the ear vein died in fourteen days. It was very much emaciated, but no other evidence of disease was discovered. Bacteria were not found in the organs or blood. All of the other inoculated animals remained well.

The discovery of *Bacillus cloacæ* in the soil is of much significance. It harmonizes the results obtained by Professor Burrill in his bacteriological examination of the roots of the affected corn with the early conclusion that the cause of the dwarfing of the corn is not primarily the result of bacteria. It also explains how it is possible for the parenchymatous tissue of the leaf sheaths, husks, and leaves to be so generally sprinkled with lesions containing this organism. Whether these organisms are of themselves able to gain entrance into the parenchymatous tissue of the leaf sheaths after being lodged by various agencies against the exterior of the plant, or whether they are inoculated into the tissues by means of insects or injuries to the epidermis otherwise inflicted is not determined. The experiments of Professor Burrill would indicate the possibility of their power to penetrate the epidermis of the leaf sheath.

It appears that this organism, which is strongly saprophytic in many of its tendencies, is capable of multiplying in the tissues of the living corn plant, and consequently may be considered a facultative parasite.

A few cultures made from similar lesions contained a total of four other species of bacteria, which evidently had survived for a greater or less time in the parenchymatous tissue of the leaf sheaths, and, to all external appearances, had produced lesions not distinguishable from those associated with *Bacillus cloacæ*. This shows the existence of other usually saprophytic bacteria capable to a certain extent of such a parasitic life. The infrequency of their appearance, however, would indicate either their existence in the surroundings in much smaller numbers, or that, on account of their strongly saprophytic nature, they are unable to survive in the tissues of the living plant. Mr. H. L. Russell[1] has recently shown that a large number of bacteria, mostly saprophytes, are able to live for certain periods of time and to spread to a limited extent in living plant tissue.

CORN SMUT (USTILAGO MAIDIS) NOT FATAL TO ANIMALS.

Although the feeding experiments made by Professor Gamgee in 1868 and those at the Illinois State experiment station more recently, described on pages 11 and 15, gave negative results, it seemed desirable to make still further tests of the etiological power of this fungus. To this end the two following experiments were made by Dr. F. L. Kilborne:

(1) The load of cornstalks affected with smut (*Ustilago maidis*) removed from the field used in a preceding experiment (page 44) were fed to three 2-year-old steers, placed separately in box stalls in a barn on Mr. McCleary's farm. The animals had been in a cornstalk field for about five weeks prior to the feeding. They were fed exclusively on the smut-laden cornstalks and free smut mixed with a small quantity of a mixture of corn meal and wheat bran for seven days without ill effects. Owing to the absence of necessary appliances the weight of the animals and of the smut fed was not taken. It is safe to say that these animals consumed a much greater quantity of smut than the animals which died in the field It was observed that the cattle did not eat the smut readily in the field, and in this experiment the animals refused it until apparently hunger compelled them to eat. The mixing of mill feed with the pure smut made it more appetizing, and consequently a much larger quantity was eaten than otherwise would have been done. The fact should be borne in mind that the smut used in this experiment came from a cornfield in which several animals had died within five days after they were turned into it.

(2) In December, 1893, six barrels of corn smut were collected from several cornfields in which cattle had not been turned, near Van Meter, Iowa, and shipped to the experiment stations of this Bureau. This material comprised both the abortive ears with the husks attacked by the smut and the masses of pure smut found growing on the stalks.

---

[1] Bacteria in their relation to vegetable tissue. Report of the Johns Hopkins Hospital, Vol. III (1893), p. 223.

The total weight was 280 pounds, of which at least one-half was smut.

Two heifers, Nos. 238 and 284, 15 to 18 months old, were placed in separate box stalls. They were bedded with peat moss instead of straw, in order that their food should consist exclusively of the prescribed ration. They were allowed to run in a small yard (16 by 30 feet) for about five hours daily.

During the experiment the animals were given all the well water they wished. They were not salted. The smut was apparently not relished by the heifers, especially by No. 238, so that it required considerable inducement to get them to eat the desired quantity.

Beginning on the morning of January 17, 1894, and continuing until noon of February 2 (sixteen and one-half days), the heifers were fed morning and evening with from 2 to 3 quarts of a mixture of equal parts by weight of cut hay and a mixture of corn meal, middlings, and wheat bran, and 16 quarts of smut. The actual quantity of the fungus consumed by No. 239 was 61 pounds, or a daily average of nearly $3\frac{7}{10}$ pounds, and by No. 284, $67\frac{1}{2}$ pounds, or a daily average of $4\frac{1}{5}$ pounds. The temperature of the animals was taken every morning and evening. The animals appeared to be perfectly well throughout the time of feeding, and continued so for several months during which time they were kept under close observation.

Table showing the temperature of animals during the feeding experiment.

Date.	No. 238.		No. 284.	
	8 a. m.	5 p. m.	8 a. m.	5 p. m.
1894.	*Degrees F.*	*Degrees F.*	*Degrees F.*	*Degrees F.*
January 16	....	102	....	102.7
January 17	101.2	101.4	101.5	102.4
January 18	101.4	101.4	101.6	102
January 19	101.2	101.8	101.5	101.4
January 20	101	101.8	102	102.2
January 21	101.4	101.6	101.8	102.6
January 22	101.4	101.8	102	102.5
January 23	100.8	101.8	101.8	102.4
January 24	100.4	101.8	101.8	102.4
January 25	100.7	101.7	102.2	102.4
January 26	100.2	101.2	100.8	101.5
January 27	101	101.2	100.7	100.8
January 28	101	102.2	100.8	102
January 29	100.7	102.2	101.6	102.2
January 30	100.8	101.4	101.7	101.6
January 31	100	102	101.6	102.6
February 1	100.6	102	101.6	102
February 2	100.4	....	100.8	....

Several masses of the smut fed in this experiment were brought to the laboratory and examined microscopically to determine whether or not there were other fungi parasitic upon or associated with the *Ustilago maidis*, and which might possess destructive properties. These examinations failed to reveal the presence of any such organism. The negative results obtained in these experiments are additional evidence of the nonetiological importance of this fungus, at least so far as its power to produce disease in cattle is concerned. Careful search was made in the cornfields in which cattle had died for any diseased condition of the corn, or for the presence of other plants or fungi which might be possessed of poisonous properties. Thus far no evidence of such a cause for the disease has been found.

ANIMALS OTHER THAN CATTLE NOT AFFECTED.

It has been of considerable interest to determine whether or not animals other than cattle are affected with this disease. Billings states that other herbivorous animals and horses are sometimes attacked. An agricultural paper, in estimating the total loss from this cause, includes a considerable number of horses. The replies to the circular letter (page 17), giving information on this disease, show that horses and sheep occasionally die while pasturing in cornstalk fields. It is a significant fact that the reports, by circular letter, of this disease in horses were for the greater part from people residing in the State of Kansas, and that Dr. Mayo[1] describes a disease[2] in horses (*Enzoötic cerebrites*, or "staggers") as occurring very extensively in the State of Kansas during the fall and winter of 1890 and 1891. He further states that there was very little cornstalk disease during this season.

In these investigations I found horses pasturing in cornstalk fields where cattle died in five of the eighteen outbreaks. Hogs fed upon the carcasses of the dead cattle in two outbreaks. Sheep were pastured in the cornstalk field in one instance. In this limited number of exposures the disease was not found in any of these animals. We have failed, therefore, to find any evidence of the existence of this disease in horses, sheep, or swine.

CHEMICAL ANALYSES.

Early in these investigations the necessity was felt for a knowledge of the chemical constituents and the physiological properties of cornstalks, especially those grown in the fields where cattle had died of this disease. Accordingly, small quantities of the following substances were collected and sent to Dr. E. A. de Schweinitz, chemist of the Bureau, for an analysis, especially with reference to the alkaloids or other substances which might be of a poisonous nature:

(1) Cornstalks, mostly leaves and tassels, from the cornfield of Mr. C. Johnson. (Outbreak No. 5, 1892.)

(2) The diseased leaf sheaths collected at Mazon, Ill., in August, 1893.

---

[1] Bulletin No. 34, Experiment Station of the Kansas State Agricultural College, 1891.

[2] This disease is said to have been caused by a fungus, *Aspergillus glaucus*, on moldy corn which was ingested by the horses. The spores of this mold are stated to gain entrance to the circulation and find lodgment in the kidneys and liver, more frequently in the latter. The spores germinate and cause inflammation of these organs. The cerebral symptoms are the result of the formation of an abscess in the brain. This is caused by an interference with the blood supply, probably from spores or mycelia of the mold in the circulation. The spores of this fungus seem to retain their infectious properties for about six months. Mayo was unable to cultivate this fungus from the organs of the dead horses.

(3) The diseased cornstalks (dwarfed) from the farm of Mr. Leon Hay, of Kankakee, Ill., which were sent to the laboratory late in September, 1893.

(4) Liver of steer No. 11, which was sent to the laboratory in glycerin in December, 1893.

For the results of these analyses the reader is referred to the accompanying report of Dr. de Schweinitz. It appears, however, that they did not reveal the presence of any alkaloidal poison or inorganic salts in unusual or abnormal quantities. The necessity for a more extended chemical investigation is indicated in the discussion on the etiology of the disease on a subsequent page.

DISCUSSION ON THE NATURE OF THE DISEASE BASED UPON DATA OBTAINED IN THE SPECIAL INVESTIGATIONS.

The data recorded in the preceding chapter permits a more comprehensive view of this disease and of its economic importance than could heretofore have been taken. Although many important facts in the natural history of this malady are yet unknown, an analytical study of the results obtained will show that this disease of cattle is not such a plague to the Western farmers as it has been depicted.

CONDITIONS UNDER WHICH THE DISEASE OCCURRED.

The apparently contradictory statements made by farmers and cattle raisers concerning the conditions under which this disease occurs are clearly explained by the diverse management under which cattle dead from this malady were found in these investigations. A review of the essential facts determined in the different outbreaks summarized in the appended table will show that the fallacy of the popular theories is undoubtedly to be found in the fact that they are based on the conditions surrounding a very few outbreaks.

A synopsis of the general conditions found in the investigated outbreaks of cornstalk disease.

Outbreak.	Locality.	Date.	Total number of cattle.	Number that died.	Length of time cattle were in cornstalk fields daily.
1	Red Oak, Iowa	November, 1892.	70	2	First 6 days, 3 hours. Continuously.
2	do	do	56	1	First 3 days, 2 hours. Continuously.
3	Ames, Iowa	do	7	2	All day; yarded at night without food.
4	Red Oak, Iowa	do	24	4	First 3 days, 2 to 3 hours. Continuously.
5	Tyner, Iowa	do	22	7	Continuously.
6	Mechanicsville, Iowa	do	37	2	Do.
7	Toledo, Iowa	do	21	3	First few days, 5 hours. Continuously.
8	Stennett, Iowa	December, 1892.	25	5	First few days, 3 to 5 hours. Continuously.
9	Wales, Iowa	do	40	3	First few days, 3 to 5 hours. Yarded at night.
*10	do	November, 1892.	15	2	Continuously.
*11	Emerson, Iowa	do	140	10	Do.
12	Woodbine, Iowa	do	33	5	First few days, 3 to 5 hours. Continuously.
13	Van Meter, Iowa	October, 1893.	35	7	Continuously.
14	New Providence, Iowa.	do	52	15	First 3 days, 2 to 3 hours. Continuously.
15	Percival, Iowa	November, 1893.	100	22	Continuously.
16	Corning, Iowa	do	75	9	Do.
17	Hastings, Iowa	December, 1893.	70	7	Do.
18	Red Oak, Iowa	do	226	18	Do.

A synopsis of the general conditions found in the investigated outbreaks of cornstalk disease—Continued.

Outbreak.	Food, other than cornstalks.	Size of cornfields.	Condition of corn.	Kind of land.	Water supply.
		Acres.			
1	Clover, meadow, and pasture.	20	Good	Upland	Spring (trough).
2	Clover meadow	About 40	....do	....do	Well.
3	None	13	....do	Upland. One-fourth acre, low and wet.	Do.
4	Clover pasture	20	....do	Upland	Do.
5	Millet and hay	12 60	Good on upland.	Small area of lowland in each field.	Stream.
6	Cut corn and tame hay.	3	Good	Upland	Well.
7	Pasture	20 30	Good on upland.	Nearly all upland	Do.
8	....do	15 20	Good	Upland	Stream.
9	None	10 20	....do	....do	Well.
*10	....do		....do	....do	
*11	....do		Fair	....do	
12	....do	20 20 20	Good	....do	Stream.
13	....do	25 20	Fair to good	....do	Spring.
14	....do	20 25 20 35	First 2 fields good, otherwise fair.	Nearly all upland	Stream.
15	....do	80	Good	Bottom land, low	Do.
16	Pasture	80	....do	60 acres upland, 20 acres, lowland.	Well.
17	None	70	....do	Upland	
18	....do	40	....do	Lowland	River.

Outbreak.	Salt given.	Time animals were known to be sick.	Quantity of smut (*Ustilago maidis*) on corn.
1	Frequently	4 to 12 hours	Less than usual.
2	Constantly in the field	Found dead	Very little.
3	Once in 5 days	1 found dead; 1 sick 2 hours	Do.
4	Once in 7 days	2 found dead; 2 sick 12 hours	Considerable.
5	Frequently	Some found dead; others sick a few hours.	Small amount.
6	....do	4 to 5 hours	None (?).
7	Salt in trough constantly	3 to 12 hours	Considerable. All picked and drawn out of field when corn was harvested.
8	None	4 found dead; and 1 sick a few hours.	Moderate amount
9	Twice a week	A few hours	Small amount.
*10	None		
*11	....do		
12	Frequently	Found dead, or sick a few hours.	Considerable.
13	....do	....do	Small amount.
14	Irregularly at first, later in field continuously.	Found dead	Do.
15		....do	Moderate quantity.
16		Usually found dead	Considerable.
17		Found dead	Do.
18		....do	Very little, much less than usual.

* At the time outbreaks Nos. 10 and 11 were reached the cattle had been dead for several days and the owners were not present. The fragmentary knowledge concerning them was obtained from other members of the families.

From the table it will be observed that in eleven of the eighteen outbreaks the animals had plenty of salt, in three instances no salt was given, and in four outbreaks this fact was not determined. The animals in each of the outbreaks had free access to an abundant supply of water, or were provided daily with well or spring water.

The cornfields in which two of the outbreaks occurred were entirely on lowland. In nine outbreaks they were entirely on upland, and in seven they were on high and low land, but for the greater part on upland. The growth of the corn was vigorous in every field on the upland, and with few exceptions the crop was a good one on the lowland. The quantity of smut on the corn varied from very little to unusually large quantities. In one instance (Outbreak No. 7) all of the smut-bearing stalks were cut and drawn out of the field at the time the corn was harvested. In most instances the cattle were allowed to remain in the corn stalks for from one to three hours only each day for the first three to six days, while in the others they were left in them continuously. In a few instances the cattle had no other food than the cornstalks, but in the majority of cases they had access to stacks of hay or straw, or were allowed to run at will over pasture or meadow lands.

A careful inquiry into the common practice of farmers in certain sections of the country where this disease occurs disclosed the interesting fact that the conditions under which the cattle were usually kept, and the methods followed in wintering them, where no deaths occurred differed in no appreciable manner from those where many animals died. It was found that farmers as a rule furnish their cattle with plenty of salt and water, and that they usually take the precaution of allowing them to remain at first for only a few hours daily in the cornstalk fields. The practice of giving other food is frequently followed. In fact, so far as I have been able to determine, the methods followed by those who lose cattle and those who do not are identical. On this account obvious and peculiar conditions, which may be considered as favoring the appearance of the disease or forming a means for its prevention, have not been discovered. The method of caring for cattle by one man who loses heavily may be followed by a neighbor whose herd remains free from the disease, although the cornfields may be on apparently the same kind of soil and separated from each other only by a fence or at most by a public highway.

It was found that in certain outbreaks the cattle were pastured consecutively in two or more cornstalk fields. In these cases the deaths usually occurred in the last field, but in a few instances very soon after transferring the animals. The time which elapsed after the cattle were turned into the cornstalks before the first death occurred was variable, though usually from five to eight days. This is of much importance, and in order to make a comparative study of this point with reference to the outbreaks investigated the appended table has been constructed:

Outbreaks.	Total number of deaths.	Number of cornstalk fields cattle were in.	Fields in which death occurred.	Number of deaths and days on which they occurred after the cattle were turned into the cornstalk fields in which they died.													
				1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1	2	1	First											1		1	
2	1	1	First				1										
3	2	1	First					1	1								
4	4	1	First								1	1		1			1
5	7	*2	First and second	†2					4					‡1			
6	2	1	First									2					
7	3	2	Second									1	2				
8	5	2	Second					3	2								
9	3	2	Second							1	1		1				
10	2	1	First					1							1		
11	5	1	First								2	3					
12	5	3	First and second		§3					2							
13	7	2	Second		1	5	1										
14	15	4	First, second, and third					‖10	1	4							
15	22	1	First						10		¶ 12						
16	9	1	First						1			8					
17	7	1	First				7										
18	18	1	First			1				17							

---

* Where the cattle were in two or more fields the time occupied in each varied from one to two weeks.

† One steer died on the first day in each field. In the first instance death resulted presumably from the effect of dehorning.

‡ The cattle were kept out of the cornfield for three days (seventh, eighth, and ninth) after the death of the four animals.

§ All in the first field.

‖ Eleven animals died in the first field. Two died in the second and two in the third field on the seventh day after being turned into them.

¶ The twelve animals died between the sixth and eleventh days.

The table shows that the great majority of deaths occurred from the fifth to the eighth day after the cattle were turned into the cornstalks. In the seven cases where the animals were in two or more fields deaths occurred in the first in three instances, and usually the time which elapsed after turning the animals into the second field, before they began to die, was as long as ordinarily occurred when they perished in the first field. This fact may be used as an argument to show that the causative agent was absent in the first, but present in the second field.

The previous history of the cattle was not obtained in all cases, but usually they had been turned into the cornstalks directly from pasture lands. As the cattle were not turned into the cornstalk fields until late in the fall it is evident that they had been feeding for several weeks upon grass more or less affected by repeated freezing.

The prevalence of the disease among young cattle was especially noticeable. Although the great majority of all the cattle in the herds in which deaths occurred were young, a few of the dairy herds consisted of young and old animals. In these the old ones invariably remained well. Those which suffered most were calves, yearlings, and 2-year-olds.

SYMPTOMATOLOGY.

As previously stated, opportunity was not afforded to observe an animal suffering from this affection prior to death. The insidious nature and rapid course of the disease usually results in the death of the animal before its presence is detected. In a few instances, however, the sick animals were observed very carefully by their owners, from whom all the knowledge we have of the symptoms was obtained. Although little dependence can be placed on symptoms as ordinarily described by unprofessional and especially interested persons, a careful questioning rendered it possible to eliminate many of the customary exaggerations and to obtain a list of the more apparent manifestations of the disease.

Mr. Bixler (Outbreak No. 1) found a steer lying down and unable to rise. His head was extended, resting on the ground and moving continually from side to side; this was kept up for several hours, when he died. In the second case a steer was found to remain behind the other animals when they were driven to water about 11 a. m. At 3 p. m. he was found in a dying condition.

Mr. Williams (Outbreak No. 4) found his animals apparently well at 5 a. m. At 7 a. m. a heifer was found resting squarely upon her knees, the head extended and resting upon the ground. She was frothing at the mouth and groaning as if in great distress and died in about two hours.

Mr. Miller (Outbreak No. 5) found a calf lying down and unable to rise. The head rested on the ground and the eyes were rolled up. It kicked considerably and appeared to suffer intensely. It was shot in a few hours to relieve its sufferings. Another calf was found sick in the morning. It was placed in a barn, where for a few hours it was furious in its efforts to get out. Later in the day it laid down and could not be induced to rise. It was quiet part of the time, but usually kicked and bellowed as if in much distress and refused all food. There was a normal movement of the bowels in the afternoon.

Mr. C. Johnson observed a steer to act as if "crazy" at night. It was found dead the following morning.

Mr. J. G. Jones (Outbreak No. 9) observed several cases. The animals were very quiet. One of them exhibited a semi-comatose condition.

Mr. Hunt (Outbreak No. 12) described the symptoms to be similar to those observed in a dog poisoned with strychnine.

Mr. McCleary (Outbreak No. 13) stated that the sick animals were dull, exhibited great weakness, and walked with a peculiar jerky movement of the hind legs. The animals trembled as if suffering from a chill. In a short time they fell down and appeared to be in great distress, heads moving continually backward and forward.

Mr. Hadley (Outbreak No. 14) observed a sick animal trembling violently for about two hours preceding death.

Although the descriptions of the actions of the affected animals as given by the owners differ somewhat, there is a striking resemblance between them. It is unfortunate that a more thorough examination, especially in reference to the temperature, pulse, and respiration, was not possible in this investigation. These important symptoms will necessarily remain undetermined until the opportunity of recording them is presented to properly trained persons..

It will be observed that the symptoms given by Gamgee do not differ materially from those here enumerated. Billings gives considerable space to the symptomatology of this disease, but for the greater part it is confined to the inoculation disease. On the evidence given we cannot at present accept the artificially produced disease as being identical with the cornstalk disease as it occurs in nature.

MORBID ANATOMY.

In all, twelve animals which died in cornstalk fields were examined. Post-mortem changes were more or less advanced in the greater number of these, although a few were examined soon after death. The post-mortems showed that while the macroscopic lesions were usually obscure and varied much in degree, they were constant and uniform in character. As would be expected, the structural changes in the various organs were very slight. This explains the statement of many veterinarians that the organs of cattle dying of cornstalk disease are normal in appearance. A summary of the lesions found in the twelve cattle examined, and their distribution in the body, is appended·

Nature and distribution of the lesions.

[Organs marked (*a*) were decomposed to such an extent that they were rejected.]

Animal No.	Outbreak.	Brain.	Lungs.	Heart.	Liver.	Spleen.	Kidneys.
*1	1	Normal	No hepatization.	Normal	(*a*)	(*a*)	(*a*)
*2	1	.....do	.....do	.....do	(*a*)	(*a*)	Pale.
3	3	.....do	Normal ...	Hemorrhages beneath epicardium.	(*a*)	Enlarged; blood beneath capsule.	Hemorrhages beneath capsule.
4	3	Hemorrhage beneath pia.	.....do	Hemorrhagic .	Enlarged; œdema about peritoneal attachment; hemorrhagic areas.	Enlarged; hemorrhagic.	Hemorrhages beneath capsule.
†5	4	....	.....do	Normal	Center of lobules deeply reddened.	Pulp soft.	Hyperæmic.
*6	5	Normal	.....do	(*a*)	(*a*)	(*a*)	(*a*)
7	8	.....do	.....do	Hemorrhages beneath serosa.	Center of lobules pale; periphery reddish.	Fibrous tufts on capsule.	Cortex pale.
8	8	.....do	.....do	.....do	.....do...........	.....do	Do.
*9	10	....	.....do	.....do	Mottled; acini indistinct.	Dark	Cortex conjested.
10	13	Normal	.....do	.....do	Acini distinct, centers reddened.	Enlarged; ecchymoses beneath capsule.	Normal.
‡11	13	Punctiform hemorrhages in meninges.	.....do	.....do	.....do...........	Ecchymoses beneath capsule.	Do.
12	18	Soft............	.....do	.....do	.....do...........	Normal...	Do.

* Post mortem changes well advanced.

† Brain not examined, as the animal was killed in the last stages of the disease by a blow on the head,

‡ Intestines were removed before our arrival.

Nature and distribution of lesions—Continued.

Animal No.	First and second stomach.	Third stomach.	Fourth stomach.	Duodenum.	Remaining intestines.	Lymphatics.
*1	Normal.	Contents very firm.	Hyperæmic, few ecchymoses.	Hyperæmic, few ecchymoses.	Normal	Normal.
*2	do	do	Hyperæmic	do	do	Do.
3	do	Normal	Normal	Normal	do	Punctiform hemorrhages.
4	do	Contents unusually soft.	Hyperæmic	Ecchymoses	Areas of congestion in ileum and cæcum.	Normal.
†5	do	Normal	do	Areas of congestion.	Areas of congestion.	Do.
*6	do	do	do	do	do	Do.
7	do	do	Areas of congestion.	do	Congested with hemorrhagic areas.	Œdematous.
8	do	do	Deeply congested.	do	do	Do.
*9	do	do	do	do	Lower ileum congested.	Normal.
10	do	do	Hyperæmic, with punctiform hemorrhages.	Normal	Normal	Do.
12	do	Contents firm.	Normal	do	do	

* Post-mortem changes well advanced.
† Brain not examined, as the animal was killed in the last stages of the disease by a blow on the head.

The table shows that the only gross pathological changes observed were those of a hemorrhagic nature. The hemorrhages were mostly confined to the serous membranes, and especially those of the heart. Ecchymoses were more or less numerous, however, beneath the pleura and over the surface of the liver and spleen. The mucosa of the intestinal tract showed areas in which the capillaries were deeply injected with occasional hemorrhage. In one case (No. 4) there were pronounced hemorrhagic areas in the heart muscle, aorta, and pleura.

The liver was frequently pale; the acini presented deep reddish centers with pale peripheries. Usually, however, post-mortem changes precluded the determination of the appearance of this viscus at the time of death.

The inner and middle zones of the lobules were more densely congested than the peripheral ones. A layer several lobules in thickness immediately beneath the capsule was usually more engorged with blood than the deeper portions of the organ. In the more densely injected areas not only the blood spaces were filled, but the liver cells were also separated from each other by a space of variable width containing blood corpuscles. In some cases there were slight changes in the appearance of the cellular protoplasm.

In all of the animals examined, in a suitable condition, the hemorrhages were of recent origin and the blood normal in its microscopic appearance. There were no discolored areas indicative of old ecchymoses. The distribution and general character of the hemorrhages, which are the most conspicuous macroscopic lesions observed, are very similar to those described in certain cases of vegetable poisoning, more particularly that of fern root.[1]

Although certain symptoms described by the owners were referable to the nervous system, lesions were not discovered in the brain or spinal cord excepting in one case (No. 4), in which there was considerable hemorrhage beneath the pia mater.

In one animal certain of the lymphatic glands exhibited more or less blood extravasation, particularly in the interlobular tissue.

Although somewhat limited in number, these examinations have shed much light upon two important lesions heretofore supposed to exist in this disease, viz, impaction[2] of the omasum (third stomach) and lung disease (pneumonia).

The third stomach (omasum) was found in one case (No. 1) to be firmer than normal. In all the others it was either normal or the contents were unusually soft.

In all of the animals examined the lungs were normal. It should be repeated that in addition to the twelve cases carefully considered, nearly as many other animals in which post-mortem changes were advanced were opened and the lungs found to be free from hepatization. The number of animals examined is enough to demonstrate the absence of pneumonia as a characteristic lesion in this disease. Furthermore, evidence to indicate a relation existing between this affection and the various forms of pneumonia to which cattle are susceptible was not discovered. This is important in view of the writings of Dr. Billings, who gives pneumonia as one of the most characteristic lesions in the disease.

Although the lesions are usually not conspicuous, the hemorrhagic changes described are readily detected upon careful examination. Unfortunately we have not the symptoms to compare with the lesions in those cases showing the less marked pathological changes. In the case in which the hemorrhages were most extensive (No. 4) the animal was reported to be sick but two hours and to have given evidence of intense suffering. The fact should not be overlooked that all of the animals from which this data was obtained had been in cornstalk fields from five to eleven days before death occurred. It will be seen from the table on page 53 that a few deaths occurred both before and after that time, but unfortunately an opportunity was not afforded of examining animals which died after such extreme exposures.

---

[1] Journal of Comparative Pathology and Therapeutics, September, 1893; also editorial, June, 1894.

[2] The popular idea of "impaction" has presumably arisen from the fact that the contents of this viscus is normally much firmer than is ordinarily supposed.

BACTERIOLOGICAL EXAMINATIONS.

A review of the results of the bacteriological examinations will show that a considerable number of bacteria were isolated from the organs of the dead animals. A careful study of these forms brought out the very significant fact that the same species of organisms was not obtained from any two of the animals, and that, with two exceptions, the same species was not found in more than one organ of the same animal. This fact renders it highly probable that the death of the animals was not due to the presence of any of the organisms isolated. The additional facts that none of these bacteria possessed unusual biological properties and that none of them were pathogenic for rabbits and mice are further indications that they were either the result of contamination from without, owing to the unfavorable conditions under which the cultures were made, or that they had migrated from some of the mucous membranes of the body to the organs in which they were found. As the greater number of the bacteria were recognized as ordinarily observed saprophytic forms it is highly probable that they were in many cases the result of external contamination. This supposition is supported by the fact that the greater number and variety of bacteria were isolated from cultures made from animals that were examined in barnyards or cow sheds with a strong wind blowing, and in the presence of several individuals who were constantly walking about, thus stirring up a considerable quantity of dust.

A more important fact in eliminating the theory of the bacterial origin of this disease is that the great majority of the tubes of culture media inoculated from the various organs and tissues of the affected animals remained clear. In all, twelve distinct species of bacteria were isolated which were not readily recognized saprophytes. Generically, there were six motile and two nonmotile bacilli, three micrococci, and one streptococcus. None of these forms produced gas in bouillon containing glucose, and they were harmless to experimental animals. The conditions under which the cultures were necessarily made were such that bacteria extremely sensitive to an exposure to a low temperature, or suited only to certain kinds of culture media, could not develop. With very few exceptions, and those unsatisfactory, no attempts were made to make anærobic cultures in the field. Agar was generally used, although a few tubes of glycerin agar, gelatin, and bouillon were inoculated with the animal tissues.

The failure to observe bacteria in the large number of cover-glass preparations and sections made from the various organs of the different animals is also evidence that bacteria discoverable by our known methods of staining were not present in the tissues. The blood cultures[1] (from two cases only), made by drawing about 20 c. c. of blood from the heart with sterile pipettes and transferring it to sterile tubes, which were exposed as soon as possible to a temperature of from 30° to 37° C., remained clear.

---

[1] This method was recommended by Dr. Billings to be very efficacious in the detection of the specific bacillus of this disease.

The failure to discover a specific or even constantly present organism in the diseased animals was paralleled by the few independent cultures made by Dr. Niles, who assisted in many of the autopsies. The fact that he made independent cultures is important, as he used culture media prepared in his laboratory, while mine was made in the laboratory of the Bureau. Although presumably a given bacillus would grow equally well in both media, the fact that media from two laboratories were employed in making cultures from the same animal shows that the failure to obtain the Billings or other organism was presumably not due to any peculiar or deleterious property which the media used by me might for any reason have possessed.

In 1892 it was impossible to make rabbit inoculations with the fresh tissues. In 1893, however, a few rabbits were inoculated with the blood and several with the brain tissue from Nos. 11 and 12. All of these rabbits remained well.

The difficulties attending the bacteriological investigation of this disease are obvious, and the use of delicate methods for the cultivation of anærobic bacteria, as well as those necessary for the study of organisms requiring a constant temperature, can not well be made in field examinations. In these investigations cultures were made in sufficiently large numbers and with the necessary care to warrant the conclusion that there is no species of bacteria which can be cultivated under ordinary conditions constantly present in the organs of cattle dying of cornstalk disease.

The negative results obtained by Professor Burrill in his examination of the Craig case in 1889 (page 15), and those of Dr. Niles from the same animals I examined are in harmony with this conclusion.

ETIOLOGY.

The varied conditions under which the cornstalk disease has been found to occur show that the existing theories concerning the cause are not in accord with the observed facts. A causal relation of corn smut to the death of the cattle can not be entertained in view of the clinical and experimental evidence that it can be eaten with impunity. The "germ theory" is likewise shown to be incapable of demonstration, and cornstalks affected with the Burrill disease have been shown to be wholesome food for cattle. The careful microscopic examination of both fresh and fixed organs and blood from a number of affected animals for the purpose of determining whether or not death might be due to the invasion of protozoa or other minute parasitic organisms, has invariably given negative results.

A careful analysis of the reports of this disease received from many interested persons, as well as the published statements already referred to, shows quite conclusively that different agencies take part in the production of the total number of the deaths annually attributed to this disease. When deaths occur within twenty-four hours after cattle are turned into the cornstalk field it is highly probable that ordinarily the cause is due to mechanical interference rather than to the effect of any specific agent, either organic or chemical in its nature. The well-known and rapidly fatal condition known as "hoven," due to the repletion of the rumen with solid food or from the evolution of gases arising from the contents undergoing certain fermentation, is presumably the cause of many of the deaths which occur within a few hours.

Dr. Going, in a paper read before the Kansas State board of agriculture in 1890, describes an enteritis in cattle produced by pasturing them in cornstalk fields late in the fall and winter. He states that the animals are sick for several days and are amenable to treatment. It is not unreasonable to suppose that many of the deaths occurring after several weeks of feeding exclusively on the dry and leached cornstalks would result in the development of a more or less severe gastritis. Veterinarians are familiar with a disease in cattle due to malnutrition and indigestion produced by the continuous feeding upon pasture grass during the winter or after it has been killed by frost. In these cases the symptoms are said to be similar to those described by Dr. Going.

The engorgement and drying of the contents of the third stomach or "impaction," is reported on good authority to occasionally cause death in cattle fed upon dry food. This condition very likely occurs more or less frequently in cattle feeding continually upon dry cornstalks. In these examinations, however, it has not been found, with one exception, and deaths from this cause are believed to be infrequent. In addition to the above there are many accidental causes, such as poisoning and traumatic injuries, which presumably occasion a few deaths that are attributed to cornstalk disease. Thus I have learned of the death of a steer which occurred within twelve hours after turning the animal into the cornstalks. This death was reported to be due to the cornstalk disease but it undoubtedly resulted from dehorning, as the hemorrhage was reported to be excessive and the weather severe.

There are, however, many deaths which can not be explained by the intervention of these known agencies or conditions, and which should be included under the popular designation of "cornstalk disease." In these investigations the animals thus affected had been in the cornstalk fields from two to fourteen days, but the greater number of deaths (see page 53) occurred in from five to eight days after the first exposure.

As already stated, the existing theories concerning the cause of the disease have not been verified, and the outcome of the observations and experiments made during these investigations indicate that the exciting cause is quite different from those heretofore announced, although the exact etiological factor has not been demonstrated.

The symptoms and clinical history, so far as obtained, and the lesions in the animals examined indicate that this disease is due to a poison.

It is evident that the determination of the nature of the toxic substance can not be made until the facts obtainable by chemical analyses concerning both the inorganic and organic constituents of cornstalks and their toxic properties upon cattle are recorded. It is furthermore not clear whether this toxic condition is due to an excessive quantity of the poisonous substance or to an unusual susceptibility on the part of certain animals. The fact should be noted that ordinarily cattle are late in the fall turned from pasture lands on which the grass has been repeatedly frozen into the cornstalk fields. This sudden change from a sparing diet to an overabundance of a single highly nutritious food would be expected to produce more or less derangement in the animal economy. The exact effect upon the animal body of the normal salts or alkaloids in the cornstalks when taken in such abundance after a period of partial fasting can not at present be recorded. The large quantity of the corn fodder stored up in the rumen during the first few days permits of the accumulation of these principles in the animal body.

The effect of different soils upon the quantity of the inorganic salts in cornstalks seems not to be fully understood. It is stated that corn has the property of taking up from the soil a large per cent of potash, which appears in the cornstalks as nitrate of potash, and which is poisonous to cattle. Mayo[1] has recently shown that cattle fed upon certain corn fodder died from poisoning with the nitrate of potash which the corn contained. He also found that cattle eating cornstalks from the same field were affected differently, some dying, while others remained well. He reports a most remarkable quantity of nitrate of potash in certain cornstalks, as the following quotation will show:

> Beneath the leaf sheath which surrounds the stock just above the joints the nitrate had crystallized in fine white crystals which resembled a white mold, but was easily recognized by tasting with the tongue. Around and in the cut ends of the stalks were solid masses of almost pure potassium nitrate. If a stalk was cut in two and tapped lightly upon the table, the crystals of potassium nitrate would be jarred loose and fall as fine powder upon the table. These stalks were grown on land adjoining a barnyard which formerly was used as a hog yard. The examination of cornstalks from a field some distance from the yard failed to reveal the existence of potash by the method previously referred to.

These observations are of much importance, as it is possible they may afford a key to the etiology of this disease. The lesions in the affected cattle reported by Dr. Mayo show much greater intestinal disturbance than those found in the animals I have examined. I have carefully examined many of the cornstalks in several of the fields in which cattle died, but have not in any instance found the presence of perceptible quantities of any crystallized substance either beneath the leaf sheath or in the stalk. This discrepancy can be readily explained by the observation that these salts appear in perceptible quantities only in corn grown on soils containing large quantities of certain fertilizers.

---

[1] Bulletin No. 49, Experiment Station of the Kansas State Agricultural College, 1895.

Another fact that points to the probable toxic nature of this disease is the recovery of certain animals (see Outbreaks Nos. 13 and 16) which showed symptoms similar to those manifested by the animals which died. It was observed by the owners of these cattle that after the crisis was passed they recovered very rapidly. Although this disease is generally supposed to be invariably fatal, it is highly probable that a large number of animals are affected and recover.

The possibility of the existence of a fatal poison in the cornstalks is shown by the report of Dr. Mayo. It is not determined, however, that potash or sodium compounds are the only substances which might be produced in unnatural proportions under certain conditions in the corn plant. As the fatal quantity of known toxic substances appears to vary much in cattle, owing somewhat to the condition under which they are administered and largely to the degree of susceptibility of the individual animals, it seems to be necessary that further chemical analyses of cornstalks should be made. The stalks should be taken under different conditions, such as late in the fall, after their exposure to the fall rains and frosts, and when grown on different kinds of soil and the physiological and toxic properties of the substances thus obtained should be tested on cattle previously kept under different conditions. As the accumulated data point to the toxic nature of this disease, that hypothesis is provisionally assumed although the exact nature of the poison is not yet determined. It appears, however, to be restricted to the corn plant, and for that reason the disease it produces is designated *Toxæmia maidis.* The theory of the toxic effect on certain animals of the normal constituents of the cornstalks when taken in large quantities is strengthened by the fact that the disease does not occur in those sections of the country where the corn stalks are cut and properly cured, and where they are subsequently fed to cattle in small quantities, but sufficient in amount to keep the animals in good condition. It is hoped that chemical investigations of this disease may clear up the doubtful points.

ECONOMIC IMPORTANCE.

It is difficult to estimate the amount of loss caused by this disease. There are no statistics from which reliable data can be obtained concerning it, and, while it occurs to a certain extent every year, it is reported to be more frequent some seasons than others. It is usually confined to one or two herds in a single neighborhood, but its distribution is irregular, and frequently townships, counties, and even larger districts in the generally infected territory remain free from its ravages for years in succession.

Dr. Billings[1] summarizes the loss from this disease by stating that "it can be safely said that this malady causes more loss in cattle to the Western farmer than all other causes combined, not excepting abortion."

---

[1] Loc. cit.

Pyle[1] reports the loss in Nebraska for the season 1889 and 1890 to be about $61,000.

The Farmers' Review for February 5, 1890, contains a summary of the reports of their correspondents concerning the prevalence of the cornstalk disease in the major portion of the corn-growing States.

The figures which are given are so divided between money value and the number of animals which perished that an accurate statement can not be made concerning either, but approximately[2] they are as follows: From Nebraska, a loss of $59,094 among cattle and $5,250 among horses. In Iowa, about 1,800 cattle and a few horses. In Missouri, about 350 cattle and a few horses; several counties are reported as sustaining a loss of from 1 to 7 per cent of their cattle. These statements show an enormous loss, but the text is not clear as to whether or not all of these animals died in the fall and winter of 1889 and 1890.

In the replies to the circular letter already mentioned, 634 men reported an ownership of 39,324 cattle, of which 3,229, or about 9.8 per cent, died in cornstalk fields in a single season.

In the twelve outbreaks investigated in 1892, 490 cattle were pastured in cornstalk fields in which the disease appeared and 46 of them died. In 1893, the percentage of loss was 14.5 per cent of the animals that were turned into cornstalk fields where the disease appeared, i. e., of the 558 animals exposed 81 died.

In estimating the percentage of loss from this disease there is great danger of an overestimate. In the township of Red Oak, Montgomery County, Iowa, there were 2,460 cattle in 1892, of which, so far as I could learn, only 6, or about one-seventh of 1 per cent of the total number, died in cornstalk fields. Further inquiry showed that in the entire county there were 29,169 (number recorded in county clerk's office) cattle, of which only 17 perished in cornstalk fields. Other years the loss was reported to be heavier, and still others less. These figures are given to show that the percentage of cattle which die in cornstalk fields is small when the thousands of herds in which the disease is not recognized, if it occurs at all, is considered.

The data obtained, therefore, show that this disease is not of such great economic importance as it has been thought. As it is noncontagious, it does not threaten the cattle industry of this or other countries to which American cattle may be shipped. In the herds where deaths occur the amount of loss is frequently heavy, but the infrequency of its appearance renders the average annual loss of cattle in any community from this disease exceedingly small. It is the universal testimony of the more experienced farmers that the amount of this disease is diminishing from year to year. Notwithstanding, it is a source of serious loss and its prevention and elimination from the country is much to be desired. To accomplish this the method of prevention about to be indicated is urgently recommended.

---

[1] Journal of Comparative Medicine and Veterinary Archives, XIII, 1892.

[2] In the reports from counties some of the statements give the number of cattle, others the value of the cattle, and still others both the number of animals and their value. From the latter, the average value of an animal was estimated and from this result the approximate amount of loss was estimated.

PREVENTION OF THE DISEASE.

The rapid course of this disease indicates that prevention is the only practical remedy. As it appears in widely separated herds, there being many localities where it has never occurred, it is evident that the practical application of any general precautionary measure must be attended with very little expense, or the burden of prevention would be heavier than the loss of the occasional animals. As the disease is not contagious measures to prevent its spread are not called for, and the adoption of any method is for the sole purpose of protecting individual farmers against a possible loss of one or more animals from this disease. It has been shown that the methods now in vogue are not trustworthy, and the adoption of those heretofore suggested would, from the nature of the disease, be impracticable.

It has been observed in these investigations that a few farmers residing in the States where the disease is most prevalent, and who cut and feed their cornstalks after the method followed in the Eastern States, never lose cattle from this disease.[1] This is further illustrated by the history of the malady. Formerly it was the custom of the farmers in the States of Ohio and Indiana to turn their animals into the cornstalk fields after the ears had been picked from the standing stalks, and they reported losing many cattle from this disease. More recently the farmers in these States, with possibly few exceptions, cut their corn when it is ripe, and subsequently feed the properly cured stalks to their cattle. Frequently these stalks constitute the food for the cattle throughout the greater part of the winter. Where this practice is in vogue the cornstalk disease has disappeared. At the present time the practice of cutting the cornstalks is becoming prevalent in the State of Illinois, and several agriculturists of that State have stated that the losses from this disease are growing less and less each year. Although these illustrations may be considered coincidences, they conform to the long experience of the farmers of the Eastern and Middle States, who invariably cut their corn and feed the cured stalks and who never lose cattle from this malady.

In view of the history of this disease and the facts brought out in the investigations, the only method of prevention which commends itself to the consideration of the farmers who wish to protect their herds from this malady is to cut their corn when ripe, cure the stalks, and subsequently feed them to their cattle. This method is based upon the experience of agriculturists as well as upon theory. The results of more scientifically arranged experiments could not be more demonstrative than those obtained by the actual experience of thousands of farmers.

---

[1] A most interesting illustration of this was found by Dr. E. C. Schroeder in the State of Missouri, in 1891. The conditions were as follows: Two farmers had large cornfields separated from each other by a wire fence. One of these men pastured his cornstalk field and lost heavily from the disease; the other cut his corn and fed the stalks with no ill effects whatever. The only difference in the conditions that could be found was in the method of feeding.

This method of prevention, if generally followed, will necessarily cause a radical change in the practice of farmers in the sections of country in which this disease occurs. At first thought it may seem impractical and unworthy of consideration, but a more careful study will show that it can be practiced with profit, especially on the smaller farms, where the amount of corn and the number of cattle are not large.

This method is urgently recommended, not only as a means of preventing an occasional loss of from 5 to 30 per cent of the cattle feeding in a cornstalk field, but also in its great economy of corn fodder.[1] The waste resulting from cattle running in cornstalk fields is notorious. The universal testimony of farmers who are feeding their cattle cut and properly cured cornstalks is that it pays. The quality of the stalks is much superior to that of the weather-beaten and bleached stalks left standing in the field. In addition to this, the amount of fodder obtained from a certain area is much increased over that utilized when the cattle run at will in the standing stalks, so that from a given acreage of corn a much larger number of animals can be wintered. While this means of prevention may not be practicable where large herds are driven from the Western plains to be wintered in the corn-growing sections where the acreage of cornstalks is extravagantly large, it is deemed practicable wherever economy in the use of forage becomes necessary. The slow but constantly increasing rapidity with which this method of feeding cornstalks is advancing from the East shows that it is a question of a few years before the custom will become universal throughout the West. When that time arrives the cornstalk disease of cattle will presumably not appear.

---

[1] The importance of cornstalks as forage has been clearly set forth from a scientific standpoint by H. J. Patterson (Agricultural Science, Nos. 7, 8, and 9, Vol. VI, 1893), who has made a chemical analysis of the separate parts of the corn plant, such as the ear, husks, tops, leaves, stubble, etc. This analysis shows that the stalks contain a greater total of food constituents than the corn itself. He also shows that there is but little difference in the digestibility of different parts of fodder and that all its compounds, except the protein, are more digestible than those of timothy hay. He shows, furthermore, that the digestible matter in the fodder alone from an acre of good corn equals the digestible matter in 2 tons of either clover or timothy hay.

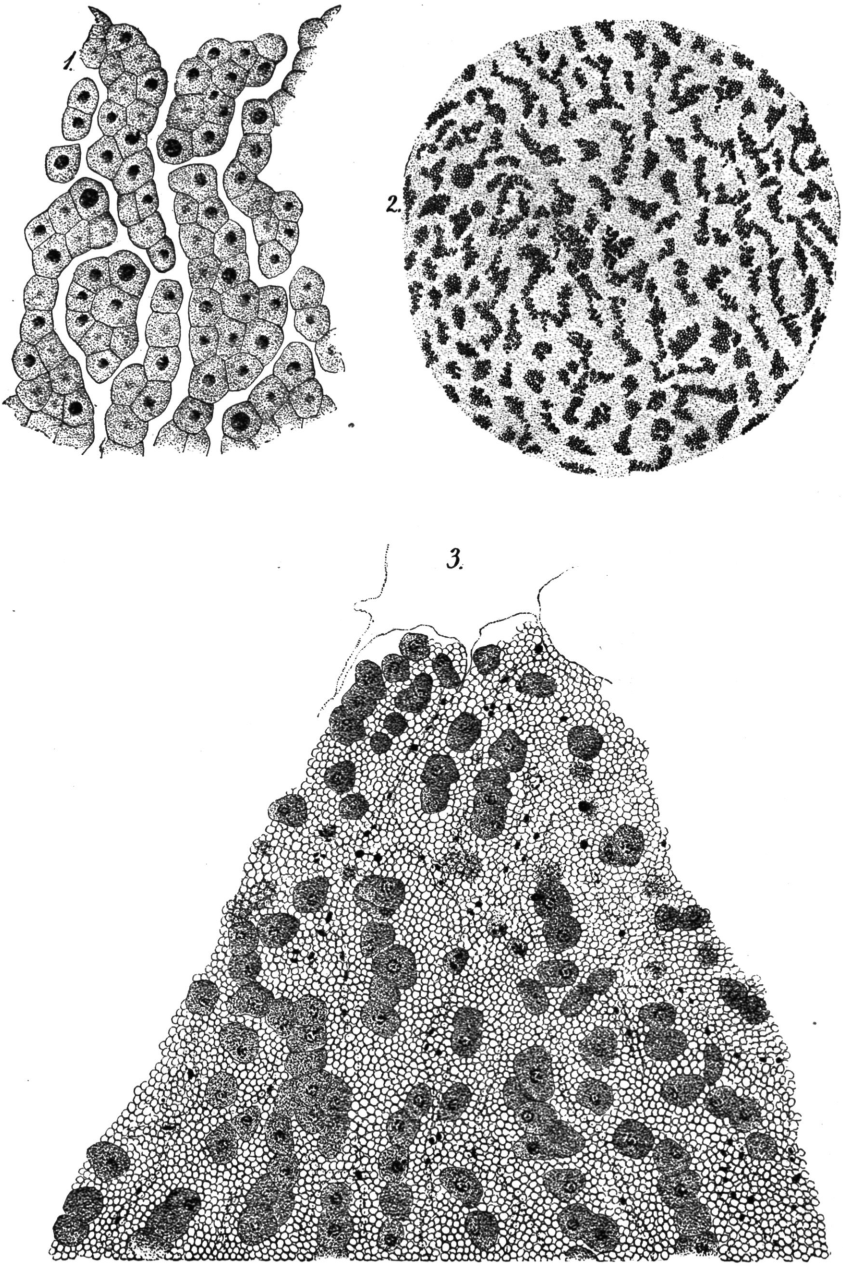
DESCRIPTION OF PLATE I.

FIG. 1.—A drawing made from a section of a lobule of the liver of a healthy cow, showing liver cells and the blood spaces (magnified 500 diameters).

FIG. 2.—A drawing from a section of liver from steer No. 4. This shows the liver cells and the engorgement of the blood spaces. Drawing traced with camera lucida, 8 mm. apochromatic objective; No. 4 eyepiece.

FIG. 3.—A drawing from a section of the liver of steer No. 3, showing the separation of the liver cells and the intercellular spaces filled with blood (magnified 500 diameters).

PLATE I.



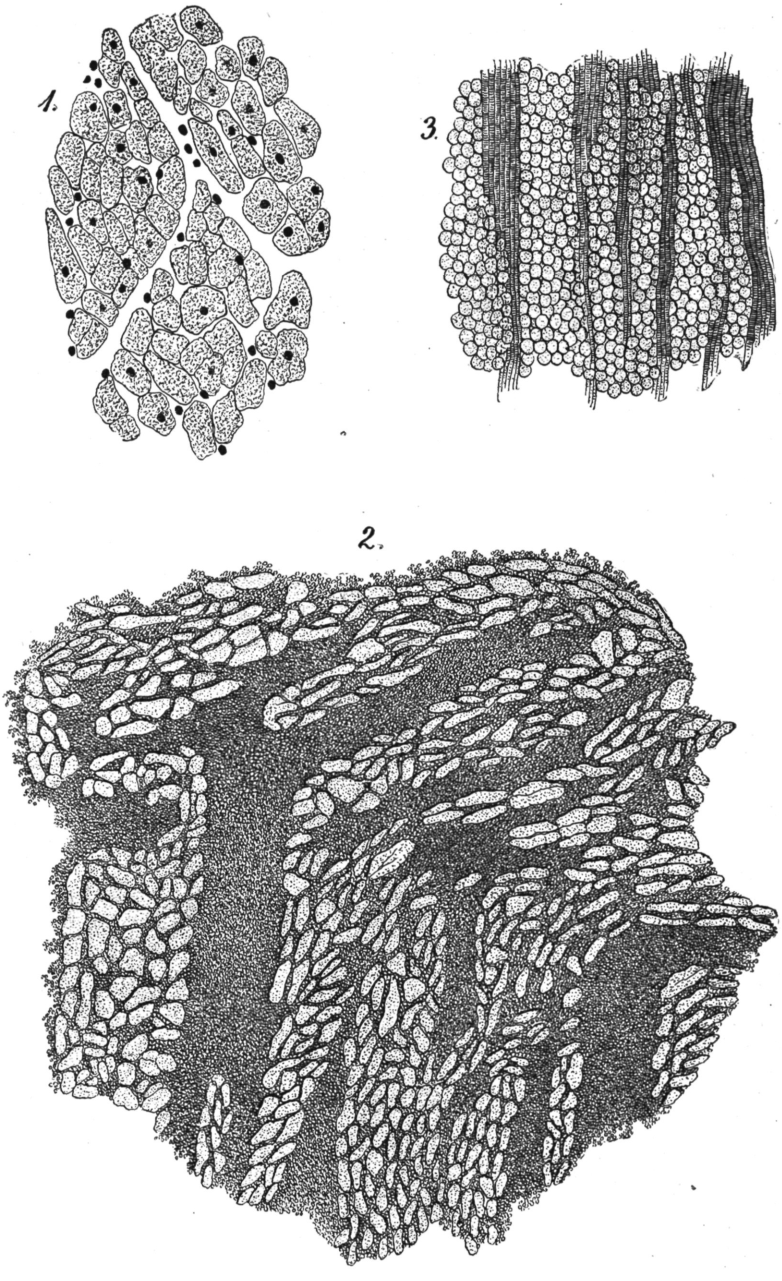
ENGORGEMENT OF BLOOD IN THE LIVER.

DESCRIPTION OF PLATE II.

FIG. 1.—A drawing from a section of normal heart muscle of a cow, showing cut ends of fibers (highly magnified).

FIG. 2.—A drawing from a section of heart muscle from steer No. 4. This shows the hemorrhage into the connective tissue surrounding the fibers. and bundles of fibers. Traced with camera lucida, 8 mm. apochromatic objective; No. 4 eyepiece.

FIG. 3.—A drawing from the same section as fig. 2, showing longitudinal fibers and bundles of fibers surrounded with blood corpuscles. The muscle fibers themselves are not affected (magnified 500 diameters).



HEMORRHAGES IN THE HEART MUSCLE.

A DISEASE IN CATTLE NOT DISTINGUISHABLE FROM RABIES.

By Veranus A. Moore.

While investigating cattle diseases in the State of Iowa in the fall of 1892, Dr. Stalker, State veterinarian, called my attention to a peculiar affection which had appeared at variable intervals during the summer among the cattle belonging to Mr. John Wade, who resided near Greene, Butler County. The mystery surrounding this malady, as well as its economic significance, rendered an inquiry into its nature and cause exceedingly desirable. I therefore visited Mr. Wade on November 10, in response to a telegram announcing the reappearance of the disease. The following history of the affection was obtained:

Early in the spring of 1892, 47 2-year-old steers were turned into a pasture containing 100 acres, the greater part of which were on slough land. The field contained several acres, however, that were dry and covered with tame grass. In the slough portion water stood for a greater part of the summer between the bogs or tufts on the tops of which wild grass grew in abundance. The cattle were salted weekly, and water was pumped daily from a well at one side of the pasture into a large trough standing in the field. The well was bored through limestone rock and was said to be about 100 feet deep.

Late in June or early in July a steer was found dead. About a month later a second animal died. Early in September a third steer was taken sick and died in about three days. October 1, 2 steers died and later in the month 4 others perished. The last 4 animals were observed to be sick for about six days prior to death.

There were two other smaller pastures on high land, in which were 25 and 27 steers, respectively. In September, 100 cattle were purchased and turned into the pastures on the upland. Early in October the surviving cattle from the slough pasture were turned into the fields on the upland with the purchased animals. They were all fed subsequently on timothy and clover hay and snapped corn. The 4 steers which died late in October had, therefore, been on high land and fed as stated above for about three weeks prior to death. None of the cattle pastured during the summer in the fields on high land had died, and all the subsequent deaths were among the cattle which had been summered on the slough pasture. Mr. Wade had not lost cattle in previous years, and to his knowledge this disease had not before appeared in his vicinity.

Mr. Wade had already lost 9 animals, and subsequently 7 others died. Of the latter 4 were examined very soon after death. The symptoms observed in the sick animals, which were similar to those said to have been manifested by the previously affected ones, and the absence of apparent lesions sufficient to cause death, suggested the probability that they were dying of rabies. As indicated by this provisional hypothesis, rabbits and calves were inoculated with the brains of 3 (Nos. 1, 4, and 5) of the 4 animals for the purpose of obtaining data whereby a positive diagnosis could be made. The results demonstrated the fact that these animals were affected with rabies, or at least with a disease not distinguishable from that malady as determined by comparative inoculation experiments. A careful inquiry was made into the prevalence of rabies in the vicinity. It was learned that about 12 miles from this place several animals, all of which died of rabies, were bitten by a rabid dog about a year before. This was the only case of the disease found to have occurred, at least for several years, in this or adjoining counties.

As the source of infection is exceedingly difficult to explain, this outbreak is of much interest. The possibility of the presence of a rabid dog can not be gainsaid. Accepting it as a fact, it is quite an unusual condition to have animals bitten by a rabid dog early in the spring, die of rabies at variable intervals throughout the summer and early winter. The fact that no other animals in the neighborhood died, that mad or strange dogs were not observed during the entire summer, and that a careful inspection of the skin of the animals examined failed to reveal the presence of scars suggestive of bites or other injuries, all tend to preclude the probability that the cattle were infected at different times by means of the bites of different dogs. The importance of this disease warrants a somewhat detailed description of each of the animals examined, and an account of the inoculation experiments resulting in the diagnosis made.

SYMPTOMS AND LESIONS IN THE CATTLE EXAMINED.

Steer No. 1.

A 2-year-old steer was found in an almost dying condition November 10. He had been sick for five days. When first attacked he was said to have been in good condition, but he was now much emaciated. Mr. Wade stated that the most marked symptoms which he observed in this animal were indifference to food and water, and persistency in driving hogs, dogs, hens, and other small animals from the yard. He would not attack persons. Occasionally he bellowed as if in great distress. The hind limbs were drawn under the body more and more each day, so that by the third day walking became difficult. At times he would run about the yard in an apparent frenzy. When observed (November 10) he walked with great difficulty. The hind legs were drawn under the body and there was marked loss of coordination. In stepping with the hind legs the weight rested first on the toes. After a few steps he would fall on the ventral surface of the body, bringing the four legs beneath him. He could rise only with difficulty. The eyes had a dull appearance and were deeply reddened. Judging from the previous case, Mr. Wade thought that he would live but a few hours. He was killed (by cutting the carotids) for examination.

Autopsy—Steer much emaciated. Mouth and œsophagus normal. Rumen contained a considerable quantity of moist, partly macerated food, consisting of straw, hay, corn, and corn husks. The third and fourth stomachs were normal. The mucosa of the duodenum and jejunum was covered with a layer of bile-stained mucus, otherwise the intestinal tract was normal. Lungs normal. There were a few subserous blood extravasations on the left ventricle. The liver was apparently slightly enlarged, normal in color. There were about 400 c. c. of a thin, clear, greenish-yellow bile.

The spleen was abnormally dark in color. The normal markings were present.

The medullary portion of the kidneys was slightly reddened. Cortex pale. Capsule easily removed. Bladder empty. The lymphatics normal.

The frontal lobes of the brain presented a surface thickly sprinkled with pigment. The blood vessels were slightly injected, especially in the choroid plexus. Spinal cord normal in appearance.

The microscopic examination, on the following day, of the various tissues did not reveal abnormal changes, and stained cover-glass preparations from the various organs and blood did not show the presence of bacteria or other microor ganisms.

Bacteriological examination.—At the autopsy tubes of agar were inoculated with pieces of the brain, liver, kidney, spleen, and the heart blood. Liquid agar was also inoculated with parts of the brain (Liborius' method of making cultures). All of the culture media inoculated with the spleen, liver, kidneys, and blood remained clear. One of the agar cultures of the brain contained *Bacillus flourescens liquefaciens* in pure culture.

November 12 an emulsion was made by grinding up a small piece of the brain tissue taken from beneath the pia of the right frontal lobe with a few cubic centimeters of sterilized water. The brain had been kept in a cool place since its removal (November 10), and when used post-mortem changes were not detected.[1] Two rabbits were etherized and inoculated with one to two drops of the emulsion, one beneath the dura and the other in the anterior chamber of the right eye. The rabbit inoculated in the eye remained well, but the other developed the following symptoms:

November 25, thirteen days after inoculation, the attendant[2] stated that the rabbit acted very queerly. November 26 he showed partial paralysis of both hind limbs and could not stand. Hyperæsthesia of the right hind leg, but the left one was apparently devoid of sensation. The rabbit tried from time to time to get up, but invariably failed. It refused food and water. There was no ferocity manifested; temperature normal. It was found dead on the following morning, fifteen days after the inoculation. The post-mortem showed injection of blood vessels of the heart. The liver was slightly reddened. Several cysticerci in abdominal cavity. Intestinal track normal. At point of inoculation the brain had a pale greenish color over an area 2 to 3 mm. in diameter. Blood vessels of the meninges slightly injected. No other evidence of disease.

Stained cover-glass preparations made from the blood and organs exhibited no bacteria. Tubes of culture media inoculated from the same remained clear.

November 28 a second rabbit was inoculated beneath the dura with a drop of the emulsion prepared from the brain of the dead rabbit. It died December 2. The examination showed an extensive imflammatory condition of the meninges of the brain about the point of inoculation.

---

[1] The delay was necessitated by the distance of the outbreak from Ames and the procuring of the rabbits.

[2] The fact that much of the time I was necessarily in the field prevented the taking of full notes on the symptoms of the animals inoculated at Ames.

STEER NO. 2.

This steer was first observed to be sick on the evening of November 10. It showed symptoms similar to those manifested by No. 1 and died November 13. It was examined November 14 in company with Dr. Niles. The post-mortem changes were considerably advanced. The intestinal tract appeared to be normal, with the exception of a thick layer of mucus over the mucosa of the duodenum. The liver, spleen, and kidneys were discolored from decomposition. A considerable quantity of clear, straw-colored serum beneath the dura. The blood vessels of the pia and choroid plexus injected. No hemorrhage. Over the frontal lobes the pia presented a stippled appearance due to fine and coarser pigment. The pigmentation was most intense about the olfactory bulbs. The microscopic examination of the pigment showed it to be composed of roundish or rectangular granules of a dark brownish color, varying from 0.5 to 1.5 μ in diameter.

Several tubes of agar were inoculated from the brain substance at the time of the autopsy. Part of these remained clear; the others developed into impure cultures of saprophytic bacteria.

As the steer had been dead for some time prior to examination rabbits were not inoculated beneath the dura with its brain. A single rabbit, however, was inoculated subcutaneously with a piece of the brain substance and remained well.

STEER NO. 3.

A 2-year-old steer was taken sick about November 10, and died during the night of November 12. He was said to have exhibited symptoms similar to those already described. He was examined November 14, but the post-mortem changes were too far advanced to determine any slight abnormal changes at the time of death.

STEER NO. 4.

A 3-year-old steer was taken sick November 19. The first symptom noticed was his persistent chasing of all the smaller animals which came near him. This was followed by paroxysms, in which he would run against fences and farm buildings. He was placed in stanchions to prevent him from injuring himself or other animals. He was at all times uneasy, would bite up a mouthful of hay or dirt and act as if in great distress. He ate and drank very little, and swallowed with apparent difficulty. He exhibited the same paralytic symptoms as described for No. 1. The feces were observed by Mr. Wade to be much darker in color than usual. He died on the morning of November 24.

Autopsy.—The examination was made within a few hours after death, the body being still warm. The animal was reported to have lost considerable in weight during the attack. The estimated weight at time of autopsy was 1,100 pounds. The lower jawbone on right side was fractured and the surrounding tissues œdematous. The mucosa of pharynx, larynx, and upper 3 inches of the trachea were sprinkled with punctiform hemorrhages which were also found in small numbers in the lower part of the trachea and larger bronchi. Considerable straw-colored œdema beneath tongue, just posterior to frænum. The lymphatic glands of the throat were deeply reddened and œdematous. Beneath the pleura on the costal surface of the thoracic cavities were numerous blood extravasations, 3 to 10 mm. in diameter. Lungs not collapsed; hyperæmic, but no hepatization in lung tissue. Ecchymoses on base of aorta and over heart. Dark post-mortem clots in both ventricles. Punctiform hemorrhages beneath endocardium over pyramids and around apex of left ventricle. Abdominal organs normal.

The brain presented no abnormal appearance, excepting a slight injection of the blood vessels. The spinal cord appeared to be normal throughout. The brain was carefully covered and placed in glycerin and sent by express to the laboratory at Washington, where rabbit inoculations were made by Dr. Smith. (See page 77.)

Bacteriological examination.—A large number of tubes of culture media were inoculated with different tissues and organs of this animal. The majority of these remained clear. A streptococcus developed in pure culture in media inoculated with the blood. It was extremely delicate in its cultural character, but mice inoculated with a pure culture remained well. This streptococcus perished before its study was completed. A spore-bearing bacillus resembling *Bacillus subtilis* was obtained from the spleen and a nonpathogenic motile bacillus was isolated from the liver.

The microscopic examination of the tissues, both in the fresh condition and in sections of the organs preserved in alcohol and Müller's fluid, failed to reveal any abnormal conditions.

STEER NO. 5.

A steer 3 years old. This animal was first observed to be affected November 21. From that time to November 24 he exhibited symptoms similar to those manifested by No. 4, excepting that the paroxysms were less marked. November 24 this animal came under my observation. He was lying down most of the time. He got up frequently and would walk a short distance and fall. In walking there was considerable trembling of the body. His movements were uncertain, the hind feet were placed well under the body and there was "knuckling" in all four feet, with an apparent loss of coordination. Temperature, 102.3° F.; pulse 42, weak; respiration, 24, not labored; temperature, 101.8° F. Smaller animals, especially dogs, were driven from the yard so long as he could walk, but no signs of ferocity observed. He would come up to a person, but when a hand was placed on his head or nose he would stop and act perfectly docile. Toward night he was unable to stand for more than a few minutes at a time. Eyes were dull and the blood vessels of the conjunctiva considerably injected. November 25 the steer was unable to rise. Conjunctiva deeply congested. Temperature, 91.6° F.; pulse very weak; respiration almost imperceptible. At 11 a. m. he was killed by bleeding.

Autopsy.—Blood appeared normal and clotted readily. No œdema or ecchymoses about mouth, pharynx, or larynx, as in No. 4. Ecchymosis in cephalic and ventral portion of lungs. Lungs not congested, but partially collapsed. A few punctiform hemorrhages beneath the endocardium of the pyramids of the left ventricle; otherwise heart normal. Lymphatics normal. Spleen much enlarged, dark colored, and friable. Upon section the capsule reverts with slight bulging of the pulp. Liver enlarged, edges rounded; in section it presented a dull, pale red color, mottled with grayish areas 1 to 3 mm. in diameter; about 700 c. c. of viscid, very dark colored bile holding solids in suspension, otherwise clear.

Kidneys normal in appearance. There were about 250 c. c. of clear acid urine in the bladder. Examination showed the absence of albumen. No lesions could be found in the intestinal tract. Contents normal.

The blood vessels of the meninges covering the brain were slightly injected. An unusually large amount of pigment on frontal lobes. Blood vessels of choroid plexus much injected. No liquid in ventricles. The gray and white matter normal in appearance.

A microscopic examination of the blood and tissues in a fresh condition showed the epithelium of the kidneys to be slightly granular. Sections of these organs from pieces hardened in alcohol and Müller's fluid showed no appreciable change in the cellular structure. The blood spaces in the liver contained a considerable number of red corpuscles.

Bacteriological examination.—At the autopsy tubes of agar and glycerin agar were inoculated with pieces of the brain, liver, spleen, kidneys, and with several loops of heart blood. The tubes inoculated with the brain, liver, and spleen remained clear. Those inoculated from the blood developed into impure cultures of saprophytic bacteria. A chromogene was obtained from the kidney. About 30 c. c. of the heart blood were transferred by means of a flamed pipette from the heart to a sterile tube. This was placed in an incubator in about eighteen hours. The serum which oozed out of the clot remained clear. Cover-glass preparations from the different organs showed, when properly stained, no bacteria or other organisms.

Anærobic cultures were made after Liborius' method from the kidney, liver, spleen, and brain. All of these remained clear.

November 28 a heifer calf (No. 19) about 2 months old was inoculated beneath the dura with a few drops of a suspension of the brain of steer No. 5. The calf was chloroformed, trephined on the left side of median line, and the emulsion injected beneath the dura by means of a syringe. A small quantity of blood and presumably a portion of the injected liquid escaped after withdrawing the needle. The wound healed nicely. The temperature remained normal for three days, after which it was not taken for a period of twelve days.[1] December 15, seventeen days after the inoculation, the temperature was 100.3° F. The calf appeared to be weak, laid down often, rose without much difficulty, and bellowed frequently. There was considerable dribbling of saliva. December 16, temperature 98° F. December 17, temperature 98.4° F. Calf was very weak, loss of coordination in walking, fell frequently, and rose only after several efforts. Some bloating. Reported by the man in charge to refuse food almost entirely. Dribbling of saliva less marked. December 18, temperature 89° F. Paralyzed. December 19, calf found dead.

Autopsy.—Calf considerably emaciated. Mucosa of mouth and pharynx normal. Lungs normal. Both ventricles contained dark, firm clots. Liver normal in appearance. About 100 c. c. of dark bile holding finely divided particles in suspension. Slight œdema in pelvis of kidneys. Urine dark, cloudy, and acid in reaction. Intestinal mucosa generally covered with mucus, a few punctiform hemorrhages in cæcum. Contents of colon firm and covered with mucus. Lymphatic glands slightly œdematous. The brain and its membranes appeared to be normal. Slight amount of pigmentation. Tubes of agar were inoculated with pieces of the spleen, liver, kidney, and brain. One of the tubes inoculated with the brain contained a few colonies of *Bacillus coli communis;* the others remained clear.

November 26 a heifer calf about 9 months old was inoculated intravenously with 14 c. c. of defibrinated blood from steer No. 5. The calf remained well.

STEER NO. 6.

This animal was taken sick December 8, and died December 12. The symptoms were reported to be the same as those exhibited by previous animals. No post-mortem examination was made.

STEER NO. 7.

A steer 18 months old died during the night of December 13. This animal was found sick December 11, and it was not known how long he had been sick. The symptoms were reported to be the same as those in previous cases. The partial paralysis and "knuckling" were especially well marked. Examination made December 14, at 10 a. m., by Dr. Kilborne.

Autopsy.—General condition good; weight about 500 pounds. Mouth, throat, and lungs were normal in appearance. Heart muscle flabby; the left ventricle nearly filled with a firm post-mortem clot. The right ventricle contained a dark clot and liquid blood. A few punctiform hemorrhages were beneath the endocardium of the left ventricle.

The liver was attached to the diaphragm by firm fibrous adhesions of long standing. The surface was marked by several scars, otherwise the liver appeared to be normal. The gall bladder contained about 500 c. c. of dark greenish viscid bile, holding solids in suspension. The spleen was enlarged to twice its normal size. Pulp of a dark blackish color, and rather soft. Malpighian bodies visible in fresh sections. The enlargement appeared to be due to an increase in the number of red corpuscles. The cortex and base of pyramids of the kidneys pale. Capsules easily removed. The bladder contained about 1,500 c. c. of clear urine. No albumen. The addition of nitric acid produced an unusual amount of effervescence.

---

[1] I am indebted to Dr. Kilborne for the notes on the symptoms and post-mortem condition.

The contents of the third stomach somewhat dry. In the fourth stomach a slight œdema of the laminæ with several erosions of the mucosa. The latter in the duodenum appeared swollen and covered with an abnormal amount of mucus. A slight injection of the blood vessels. In the ileum the mucosa was covered with a thin layer of cells. Cæcum and colon normal. Lymphatic glands normal in size. The membranes covering the brain presented an arborescent appearance from the injection of the blood vessels. A slight hemorrhage on the left hemisphere near the longitudinal fissure. Considerable pigment over the frontal lobes. No fluid in the left lateral ventricle. A small quantity of blood-stained serum in the right lateral ventricle. Choroid plexus deeply injected.

A microscopic examination showed the blood and organs to be apparently normal. Tubes of culture media were inoculated with pieces of the liver, spleen, and kidney. Those inoculated from the spleen and kidney remained clear. One of the agar tubes inoculated with the liver developed into a culture of a motile bacillus. Unfortunately this culture was inadvertently rejected.

COMPARATIVE INOCULATIONS.

The positive diagnosis of rabies was not made until the results of a series of inoculations with the virus obtained from steer No. 4 were obtained and carefully compared with those of a parallel series with virus obtained from a rabid dog. As previously stated, the brain of steer No. 4 was carefully removed, placed in pure glycerin, and shipped by express to the laboratory at Washington, D. C., where it was received four days later. It was kept in a refrigerator for five days, when Dr. Theobald Smith made, from a portion of the medulla, a suspension in peptonized beef broth and injected about 0.24 c. c. beneath the dura of rabbit No. 398. This rabbit died in about seven weeks with symptoms of paralytic rabies. Two rabbits inoculated with an emulsion prepared from a piece of the brain of rabbit No. 398 subsequently died with similar symptoms. The series of inoculations thus begun extended over a period of nearly seven months, in which eleven rabbits were used, all of which died with symptoms of paralytic rabies.

Although comparative inoculations were made in several rabbits and calves which were carefully watched during the disease and examined post-mortem with the greatest care, there was not present any constant condition or conditions which enabled one to detect a difference between the diseases produced by the virus from different sources. The essential data obtained in these inoculations are here given.

INOCULATIONS WITH THE VIRUS ORIGINALLY OBTAINED FROM STEER NO. 4.

Rabbits and calves were inoculated with this virus beneath the dura, intravenously, and into the subcutaneous tissue. The data important from the diagnostic standpoint was obtained from the intracranial inoculations. The other methods were usually ineffective, although a few fatal results were obtained by the subcutaneous process. Atten-

tion is called particularly to the symptoms manifested by the different rabbits and the uniformity in the time between inoculation and death. The long period of incubation in the first rabbit inoculated was presumably due to the length of time the brain remained in the glycerin prior to its use. The number of rabbits used for subdural inoculation, the date of inoculation and death, are given in tabulated form. The symptoms and post-mortem notes for each animal follow the table.

A.—*Subdural inoculations in rabbits.*

Rabbit No.—	Virus used.	Date of inoculation.	Date of death.	Period of time rabbits lived after inoculation.
				Days.
398	Brain of steer No. 4	Dec. 3, 1892	Jan. 18, 1893	46
420	Brain of rabbit No. 398	Jan. 18, 1893	Feb. 6, 1893	19
418	....do	.....do	.....do	19
424	Brain of rabbit No. 420	Feb. 9, 1893	Mar. 5, 1893	24
433	Brain of rabbit No. 424	Mar. 9, 1893	Mar. 31, 1893	22
434	....do	.....do	Mar. 30, 1893	21
458	Brain of rabbit No. 434	Mar. 31, 1893	Apr. 19, 1893	20
496	Brain of rabbit No. 458	Apr. 21, 1893	May 9, 1893	18
9.	Brain of rabbit No. 496	May 10, 1893	May 29, 1893	19
449	Brain of rabbit No. 9	June 3, 1893	June 24, 1893	21
56	Brain of rabbit No. 449	June 26, 1893	July 18, 1893	22

RABBIT NO. 398.

This rabbit remained well until January 16, forty-three days after its inoculation, when it became partially paralyzed in the hind limbs. There were no other signs of brain derangement. The rabbit ate sparingly of green cabbage.

January 17: The rabbit was found lying on one side feebly moving the forelegs, gasping as if dying. This condition continued all day.

January 18: Rabbit found dead. Subcutaneous veins filled with tarry blood; marked venous injection of the intestines. Cortex of the kidney and the base of pyramids pale, the remaining portion of a deep reddish color. Liver pale; spleen barely enlarged. Lungs pale. Heart contained tarry blood. The brain surface was marked with injected blood vessels.

RABBIT NO. 420.

Paralysis observed February 2, fifteen days after inoculation. It was unable to move its hind limbs, but dragged itself along with the forefeet.

February 3: Rabbit sat in its cage with both hind feet extended backward. Its eyes had a peculiar staring look. It smelled of food when presented, but refused to eat.

February 4: The symptoms were aggravated since yesterday.

February 5: Rabbit on one side breathed regularly.

February 6: Found dead in the morning. Membrane of the brain slightly injected; the gray and white matter normal in appearance; ventricles normal. Spinal cord not visibly affected. Lungs hyperæmic. Heart normal in color. The liver very dark; a microscopic examination showed a large amount of pigment. Spleen and kidneys normal. Slight venous injection of intestines. Tubes of agar inoculated from the various organs remained clear. Cover-glass preparations showed no microorganisms.

RABBIT NO. 418.

Exhibited the same symptoms as rabbit No. 420. The post-mortem showed a marked hyperæmia of the kidneys, otherwise not different from No. 420.

RABBIT NO. 424.

March 2, twenty-one days after its inoculation, this rabbit was paralyzed in the hind limbs; could barely drag himself a few inches by using forefeet. Ate very little. Hitherto he appeared to be perfectly well.

March 3: Rabbit could not use his hind feet; otherwise appeared to be better than yesterday.

March 4: Rabbit was sitting up in usual position; paralysis much improved, but appeared to be in distress.

March 5: Rabbit found dead. The liver was very dark, containing pigment. Heart muscle unusually pale. Lungs slightly congested. The brain appeared to be normal

RABBIT NO. 433.

March 29: Twenty days after inoculation this rabbit appeared to be dull, but when placed on the floor would hop about slowly.

May 9: Head drawn back. Respiration slow. Died about 2 p. m. Mouth and fauces normal. Stomach contained partially digested food. Liver deeply reddened; contained much blood and pigment. Spleen normal. Kidneys pale, parenchymatous degeneration of cortex. Heart muscle unusually large; blood vessels injected. Lungs hyperæmic. The brain appeared to be normal.

RABBIT NO. 9.

This rabbit exhibited its first sign of the disease on the sixteenth day. Its temperature was taken daily at 8 a. m. For the first sixteen days it was normal excepting on the ninth, twelfth, and fifteenth days, when it reached 104.8°, 104°, and 104.2° F., respectively.

May 26 (sixteenth day): Temperature 102.2° F. Rabbit had difficulty in using hind legs.

May 27: Temperature 101.5° F. Partial paralysis of hind legs in the afternoon.

May 28: Temperature 89.8° F. Lay on one side, but was able to move all four feet. Respiration slow but easy. Head was drawn slightly backward.

May 29: Rabbit unconscious. No reflex movement when touched. Respiration very slow. It died about 11 a. m. Mouth and pharynx normal. The blood vessels of the trachea slightly injected. In cephalic lobe of right lung there was a small necrotic area. The blood vessels of heart slightly injected. Lungs hyperæmic. Liver dark, containing much pigment. Kidneys pale, especially cortex. Brain normal in appearance.

RABBIT NO. 449.

Fifteenth day: The temperature on the ninth day was 104.8° F. On the following day it was 103.8°; subsequently it was normal. The first symptom noticed was a marked inclination to sit close in one corner of the cage and to refuse to move.

June 20 (seventeenth day): Rabbit considerably excited; respiration accelerated. A noticeable loss of coordination in the hind limbs. Temperature 102.8° F. In the evening rabbit lay on one side unable to rise. When touched it moved its forefeet slightly.

June 21: Rabbit unable to move its feet. The head moved a little. Respiration easy. In the evening a slight tapping on its cage induced a feeble movement of the head. Otherwise paralyzed.

June 22: Rabbit remained the same as yesterday.

June 23: Respiration labored. A very slight movement of the head when the cage was tapped. A few movements of the lower jaw noticed.

June 24: No change from yesterday. When turned over a slight movement of the head noticed. Head drawn backward. Rabbit died about 6 p. m. Liver deeply reddened. Brain normal in appearance.

RABBIT NO. 434.

This rabbit remained well for several days, when it showed signs of paralysis.

March 29: Rabbit appeared dull, moved only after much urging, and had very little use of hind legs. It failed rapidly during the morning. At 11 a.m. it was barely able to sit up; hind legs projected backward. At 4 p.m. it lay on one side; no movement of body visible, except slow respiration and occasional twitching of forefeet and nose.

March 30: Rabbit found dead in the morning.

The mouth and fauces normal in appearance. The liver much enlarged and engorged with blood. Examination showed much pigment. The spleen slightly enlarged and very dark colored. Heart muscle pale. Lungs hyperæmic. The blood vessels of the brain injected, giving the gray matter a faint pinkish tint. Ventricles normal in appearance.

RABBIT NO. 458.

Rabbit appeared to be well for fourteen days. It then exhibited marked emaciation and scratched the bottom of its cage almost continuously. These symptoms continued for four days, when the rabbit was found lying on one side as if in a semi-comatose condition. A little later it was observed climbing about the cage and could walk without falling. A few hours later it was lying on one side. When disturbed it moved all four feet, but made no other effort to rise. Two hours later it was apparently unconscious. When touched with a stick it moved the forefeet very little and lay on one side as before. No visible change on the following morning, except that the respiratory movements were much more feeble. It died early in the evening.

Mouth and fauces normal. Blood vessels of heart and lungs hyperæmic. The liver very dark, containing much pigment. Brain appeared to be normal.

RABBIT NO. 496.

This rabbit remained well for fourteen days. May 5 (the fourteenth day) it sat in one corner of its cage. When disturbed it showed slight nervousness, and if forced to move was uncertain in its steps; toward night movements became difficult—unable to walk naturally.

Mouth and pharynx normal. Liver deeply congested. Brain normal in appearance, except that at the point of inoculation there was a yellowish necrosed area about as large as a pin's head. No other lesions.

RABBIT NO. 56.

On the twentieth day after inoculation the rabbit exhibited peculiar movements when disturbed; on the following morning it was partially paralyzed in the hind limbs. In the afternoon paralysis became complete in all four limbs. It lay on its abdomen with the head turned to the left. It remained in this position for about twenty-four hours, when it died. Liver very dark, otherwise organs appeared to be normal.

B.—*Inoculation of cattle.*

Animal number.	Virus used.	Method of inoculation.	Date of inoculation.	Date of death.	Time from inoculation until death.
Calf No. 19*	Brain of steer No. 3	Beneath dura	Nov. 20, 1892	Dec. 19, 1892	29 days.
Calf No. 239†	Brain of rabbit No. 424.	Subcutaneously right side of neck, 2 c. c. of emulsion.	Mar. 7, 1893	Apr. 6, 1893	30 days.
Yearling No. 241	Suspension, brain of rabbit No. 424.	Beneath dura, 0.10 c. c.	do	Mar. 26, 1893	19 days.
Yearling No. 242	do	do	do	do	19 days.
Calf No. 238	Filtrate, brain suspension, yearling No. 242.	do	Mar. 27, 1893		Remained well.
Yearling No. 238	Suspension, brain of yearling No. 242.	do	do	Apr. 14, 1893	18 days.
Yearling No. 249	Suspension, brain of rabbit No. 436.	Subcutaneously 0.3 c. c.	May 10, 1893		Remained well.

* For notes see page 76.

† The calves were from 6 to 8 months old.

YEARLING NO. 241.

Female. Inoculated beneath dura with 1 c. c. of an emulsion of the brain of rabbit No. 424, March 7. She remained apparently well for fifteen days. March 22 she refused food and water. She was very uneasy, pressed her head vigorously against the sides of the stall, bellowed very frequently, more especially when a person was in sight. At times she pawed and stamped with the forefeet. On opening the door of the pen she made vigorous efforts to escape and did not notice either words or whip. In the yard she followed a person, pressing her nose forcibly against him, but not viciously. The bowels had been loose for two days (this was thought to have been due to the food given). Temperature, 103.8° F., taken every day at 8 a. m. Slight dribbling of saliva.

March 23: She ate and drank very little; temperature 100.3° F.; considerable diarrhea. Very uneasy; bellowed frequently. No rumination.

March 24: Temperature 102° F. Animal continued to press her head against the side of the stall, bellowing almost continuously. The presence of a dog produced no effect. Diarrhea continued; considerable tenesmus. She laid down frequently, often rose immediately, but at times would lie quietly for some minutes.

March 26: Temperature 99° F. Animal lying down, unable to get up. She died about 4 p. m.

Autopsy made March 27.—Post-mortem changes somewhat advanced. The intestinal tract appeared to be normal, excepting a few punctiform hemorrhages in pyloric portion of the stomach. Spleen normal. Liver and kidneys slightly discolored from decomposition. No lesions determined. Hypostatic congestion of right lung. Considerable œdema about the heart. There were about 400 c. c. of a clear, dark, wine-colored liquid in the pericardial sac. No hemorrhages in the heart muscle, which was of a bluish color. Ventricles empty, dark blood clots in both auricles. The brain was somewhat soft, apparently due to post-mortem changes, otherwise normal in appearance.

CALF NO. 239.

Female. The animal remained apparently perfectly well until April 1, twenty-four days after its inoculation. On that day it refused all food. Small drops of perspiration had collected thickly on the hairs on the dorsal aspect of the body. Each breath was accompanied by a slight moan. The entire body in a tremble. Temperature 104.5° F., which hitherto has been normal.

April 2: Temperature 103.2° F. Refused food. Bellowed frequently without apparent cause. When approached she presented a surprised appearance, head elevated. In the evening she followed other calves, frothing at the mouth.

April 3: Temperature 103.8° F. Trembling of the entire body; stood with head erect, bellowing occasionally. When walking showed a marked loss of coordination in the hind legs, with unsteadiness in forefeet; considerable swaying from side to side. The forefeet were placed outward in a bracing position. Later in the day walking became impossible and she lay on one side, unable to get up.

April 4: Temperature 100.5° F. Lay on one side, with legs extended and head drawn backward. When approached slight movement of limbs and faint bleating; refused food, but drank about 3 quarts of water. Temperature in evening, 98.2° F.

April 5: Temperature 96.2° F. Lay perfectly quiet; no movement of body; raised head slightly when approached. Refused all food and water. Offered no resistance in being rolled over. Head drawn backward much more than normal. No retraction of the eyeballs; no trembling or twitching of the muscles. Toward night there was apparently complete paralysis.

April 6: Calf found dead early in the morning.

Autopsy.—Weight about 250 pounds. Condition good. The calf has lost very little flesh during sickness. When skinned considerable dark blood oozed from the cut ends of the veins. The spleen was much enlarged and dark colored, the normal markings nearly all obliterated. The blood vessels in the capsules injected. The liver was slightly enlarged. Capillaries filled with blood, which flowed freely upon section. Many of the liver cells in a state of fatty degeneration. The gall bladder contained about 100 c. c. of thick greenish, slightly viscid bile holding flaky mucus in suspension. A slight congestion of the cortex of the kidneys, tubular epithelium granular, otherwise normal. Lymphatics normal in appearance.

The mouth, pharynx, larynx, and œsophagus normal. The rumen contained considerable rather dry food. The omasum contained a normal quantity of food. The abomasum was empty, and the mucosa, excepting in the pyloric portion, deeply reddened. The mucosa of the duodenum deeply reddened with a few hemorrhagic areas and the contents blood stained. The mucosa of the jejunum and ileum sprinkled with small hemorrhagic areas. Contents blood stained. Cæcum and colon nearly empty, the mucosa covered with a thin layer of whitish somewhat viscid mucus.

The trachea and bronchioles contained very little mucus. The cephalic lobes emphysematous. The entire lungs had a bluish red color due to capillary injection. Upon section a small quantity of blood-stained serum oozed out of the cut vessels. A few punctiform hemorrhages on the pericardium. In left ventricle and auricle were firm dark clots which extend into the large blood vessels. In right side of heart were small clots also extending into larger blood vessels. The brain showed considerable pigment over frontal lobes. The blood vessels injected, especially in choroid plexus. Upon section small drops of blood oozed from the cut vessels. Ventricles normal. In certain of the cells from the gray matter peculiar highly refractive bodies were observed in the cellular protoplasm, outside of the nuclei. They were not observed to move in the myelinic fibers. In a few instances these bodies appeared to move. The spinal cord seemed to be normal.

A large number of tubes of agar were inoculated with brain tissue from various parts of that organ and with pieces of the other organs of the body. In one tube inoculated with the blood a few cultures of a streptococcus were obtained. It possessed no pathogenic effect on mice. All of the other inoculated tubes remained clear.

YEARLING NO. 242.

Inoculated with an emulsion of a bit of the brain of No. 241. It manifested precisely the same symptoms as No. 241, but died about sixteen hours sooner. Its temperature varied from 100.8° F. to 103° F. for the first fifteen days. The last four days it was 102.6°, 103.8°, 103.8°, and 103.6°, respectively. The autopsy showed lesions similar to those found in No. 241. No bacteria were discovered in the organs.

CALF NO. 247.

A heifer about ten months old. Inoculated March 7. She remained well until April 7, when she refused food; otherwise no symptoms noticeable until April 11, when she had paroxysms of bellowing, and hooked and pawed her bedding considerably. Upon being approached she manifested a marked desire to press her nose against the individual. Temperature normal. She drank occassionally from a bucket of water in the stall. These symptoms continued for two days without change. There was a rapid loss of flesh.

April 13: In the morning she was noticed to froth at the mouth slightly and to be much more restless; bellowed frequently, but was not disturbed by the presence of a person. She occasionly drank a few swallows of water. Temperature 103.5° F. The eyes were normal in appearance. Considerable perspiration.

April 14: Heifer found dead in the morning. Examined about 2 p. m. The post-mortem changes considerably advanced. Areas of deeply reddened mucosa in the true stomach and intestines, especially cæcum and upper colon. Considerable whitish froth in trachea and bronchioles. Lungs not collapsed. About the base of the heart and along ventricular groove were many petechiæ. Large, firm blood clots in both ventricles and auricles extending into the larger blood vessels. No lesions observable in the brain. The gray matter was somewhat softened, due presumably to post-mortem changes.

INOCULATION OF RABBITS AND A CALF WITH THE BRAIN OF A RABID DOG.

The virus for this series of inoculations was obtained from a rabid dog brought to the experiment station by Dr. Grenfell, of Washington, D. C., March 22, 1893. It had been affected for about three days and was killed the day following its arrival. A calf and two rabbits were inoculated by Dr. F. L. Kilborne with a suspension prepared by grinding a small portion of the medulla with a few cubic centimeters of sterilized bouillon. The calf and one rabbit were inoculated beneath the dura; the other rabbit was injected subcutaneously. In addition to these inoculations several others were made for diagnostic purposes from the brains of dogs suspected of rabies, or reported to be suffering from it. A considerable number of these died after exhibiting symptoms similar to those produced in animals after their inoculation with the brain of the dog first mentioned and which are recognized as diagnostic for rabies.

The more important data obtained in these inoculations are recorded in tabulated form, followed by a detailed account of the symptoms, and the lesions found in the different animals on post-mortem examination:

Inoculation of animals with the brains of rabid dogs.

Animals.	Virus used.	Method of inoculation.	Date of inoculation.	Date of death.	Period of inoculation.
					Days.
Rabbit No. 448	Brain of rabid dog No. 1	Beneath dura	Mar. 24, 1893	Apr. 6, 1893	13
Rabbit No. 447	do	Subcutaneously	do	Apr. 20, 1893	27
Rabbit No. 495	Brain of rabbit No. 447	Beneath dura	Apr. 21, 1893	May 2, 1893	11
Rabbit No. 481	Brain of rabid dog No. 2	do	Apr. 11, 1893	Apr. 28, 1893	17
Rabbit No. 50	Brain of rabid dog No. 3	do	June 20, 1893	July 8, 1893	18
Calf No. 246	Brain of rabid dog No. 1	do	Mar. 24, 1893	Apr. 3, 1893	10

RABBIT NO. 448.

Inoculated beneath the dura with about 0.2 c. c. of an emulsion of the brain of a rabid dog, March 24, 1893.

April 4: Rabbit sat in one corner of its cage. When disturbed its skin quivered and it appeared to be excited; apparent loss of coordination; refused fresh green food.

April 5: Rabbit quiet. When disturbed it moved with difficulty. Eyes partially closed. It was rolled over on one side, when it struggled feebly to get up, but failed.

April 6: Rabbit paralyzed. Lay stretched out at full length, apparently dead; respiration very slow. It died later in the day.

Autopsy showed deeply reddened liver; blood vessels of the brain slightly injected; no other lesions.

RABBIT NO. 447.

Inoculated subcutaneously with a suspension of brain of rabid dog (same as rabbit No. 448), March 24.

April 21: Rabbit found dead. Was not observed to be sick prior to death.

Autopsy showed a deeply reddened liver, due to blood stasis. Lungs partially collapsed. Brain anæmic. In order to determine the presence of rabies in this rabbit its brain was used for a subsequent inoculation which produced characteristic symptoms of rabies and fatal results.

RABBIT NO. 495.

This rabbit remained well for eight days.

April 29: It exhibited unsteadiness in walking. Temperature 103.8° F.

April 30: Loss of coordination, especially in hind legs. Temperature 102.6° F.

May 1: Rabbit lay paralyzed on the bottom of its cage. Respiration rapid; pupils dilated and head drawn backward; later in the evening slow respiration was the only sign of life. Temperature 90° F.

May 2: Rabbit died about 7 a. m.

Autopsy.—Considerable viscid mucus in small intestines. In the ileum several small ulcers. Liver pale, very fatty. Blood vessels of the heart slightly injected. Mouth and fauces normal. Blood vessels of the brain slightly injected.

RABBIT NO. 481.

This rabbit was inoculated with the brain of rabid dog No. 2.

April 27 (sixteen days after inoculation): The rabbit showed signs of paralysis in the hind legs early in the morning, but late in the afternoon the paralysis was nearly complete; respiration slow.

April 28: Complete paralysis. Very slight and slow respiratory movements. Rabbit died about 3 p. m.

Autopsy.—Much mucus in the small intestine. Liver very dark and contained much pigment. Mouth and pharynx normal in appearance. Blood vessels of the brain somewhat injected.

RABBIT NO. 50.

This rabbit was inoculated with the brain of rabid dog No. 3. No symptoms observed until July 6 (sixteen days after inoculation), when it appeared to be uneasy; later in the day there was marked loss of coordination in the use of the hind legs.

July 7: Rabbit lay on the floor of the cage, unable to rise. Slight twitching of muscles when disturbed.

July 8: Complete paralysis. Respiratory movements barely perceptible. It died late in the afternoon.

Autopsy.—Liver dark, otherwise organs appeared to be normal.

CALF No. 246.

This animal was inoculated beneath the dura with a suspension of the brain from rabid dog No. 1, March 24, 1893.

April 2: Calf was uneasy, eyeballs retracted, considerable frothing at the mouth. When approached it bellowed and endeavored to escape from the pen, seeming to be frightened. Temperature 101.8° F.

April 3: Calf became much excited while taking its temperature, which was accomplished after much difficulty, 105.4° F. being recorded. Calf had symptoms of convulsions; laid on one side; considerable trembling of muscles, and the head drawn back. Later it became partially paralyzed. Evacuation of bowels normal. It died early in the afternoon.

Autopsy.—Weight about 275 pounds; condition fairly good. Intestinal tract apparently normal throughout. Heart muscle sprinkled with a few punctiform hemorrhages. Dark blood clots in each of the heart chambers. Petechiæ beneath pleura over both lungs, considerable frothy mucus in the trachea and bronchioles. At the point of inoculation in the brain there was a slight necrosis of tissue, blood vessels slightly injected, otherwise brain and spinal cord normal in appearance. A rabbit inoculated with the brain from this calf died of rabies on the twelfth day subsequently.

The foregoing notes on the manifestation of the disease produced by the virus obtained from steer No. 4 in the Greene outbreak and from the brains of rabid dogs in the District of Columbia show that the inoculation diseases were identical. It has already been shown that the disease produced in rabbits with the brain of the steer was readily communicated to cattle by inoculation with the virus obtained from the affected rabbits, and, furthermore, the symptoms of the inoculated disease in the calves were similar to those manifested by the cattle which died in Mr. Wade's herd. A study of the symptoms shows that partial paralysis was usually the first indication of the disease. The average length of time which elapsed from the appearance of the first symptoms until death occurred was practically the same in both instances, but the average period of incubation was appreciably shorter in the rabbits inoculated with the dog virus.

The post-mortem appearances were practically the same in all cases; at least the differences observed in two animals inoculated with the same virus were quite as marked as those observed in rabbits inoculated with the virus from the different sources. It is important to note that the mouth, larynx, and intestinal tract usually presented a normal appearance. The enlarged and deeply reddened condition of the livers which contained much pigment was the only constant lesion observed.

The positive diagnosis of rabies, or a malady not distinguishable from it, in this outbreak of cattle disease is of much importance. As the animals were not furious the possibility of rabies was not entertained by the owner who had witnessed the furious form of the disease produced by the bite of rabid dogs.

This determination is furthermore significant in throwing light upon the probable nature of other outbreaks of a somewhat mysterious disease among cattle occasionally reported in the Western States. The most serious of these on record is an outbreak which occurred in a herd of cattle pasturing on the bottom lands along the Des Moines River near Emmetsburg, Iowa, in 1891. Dr. Stalker tells me that the symptoms manifested and the post-mortem appearance of the affected animals in the two outbreaks were similar in every particular. Unfortunately rabbits were not inoculated with the brains from the animals examined at Emmetsburg. It is interesting to compare the conditions under which the two outbreaks occurred. In both cases the presence of rabid or strange dogs was not known. The cattle in the two affected herds were on lowland, better known, perhaps, as "slough". pasture. Although Mr. Wade's cattle were furnished well water, it is highly probable that the animals drank more or less of the stagnant water which remained throughout the season in pools between the bogs, as they were never driven from the lowlands to the watering trough. In the affected herd at Emmetsburg it is stated that the cattle were compelled to drink exclusively from a pond of stagnant water. It is a curious and most interesting fact that there were two other herds of cattle on the bottom lands of the Des Moines River at the same time, but they were driven daily to the river for water, and these two herds remained free from the disease. While the known facts in these outbreaks in no way demonstrate their identity, they suggest a striking similarity between them and indicate a possible relationship between the conditions under which the cattle were kept and the disease.

Until the specific cause of rabies is demonstrated we are able to recognize the virus in the brain substance of the affected animals only by its ability to reproduce the disease when properly inoculated into other animals. As shown in the inoculation experiments, the filtrate (through a bougie of a Pasteur filter) of a suspension of the brain substance of a rabid animal would not produce the disease, while the injection of the suspension was followed by characteristic symptoms and fatal results. This fact, which confirms the results of other investigators, indicates that the specific cause is not a soluble substance. The extensive bacteriological investigations made by several European workers indicate that the virus of rabies is not bacterial in its nature, and the presence of other microscopic organisms have likewise not been demonstrated. Several interesting facts concerning the nature of rabies were noted during the course of this investigation, but as they do not pertain to the diagnosis of the disease in this outbreak among cattle they are withheld for a future publication.

The results of this series of investigations indicate that rabies, or a closely related disease, sometimes occurs among cattle kept under certain conditions without the intervention of rabid dogs or other animals. A review of the voluminous literature on rabies shows that many supposed sporadic cases are recorded, and also considerable evidence that the bites of several species of animals have produced the disease. Until the specific cause of this malady is determined it is suggested that cattle raisers should consider the possibility of its occurrence when they turn their cattle upon low, boggy pasture land.

Although a remedy is not known it is quite probable that if the animals are removed to high land as soon as the first deaths occur the amount of loss will be much less than if they are allowed to remain on the lowland throughout the season, especially when the disease appears early in the summer. This is indicated by the history of the outbreaks cited. Mr. Wade's cattle were removed early in the fall and the loss was only about 30 per cent, while at Emmetsburg it is stated that the cattle were not taken up until late in the fall, and the loss was very much greater.

LETTER OF SUBMITTAL.

SIR: I herewith submit the result of the examination of cornstalks supposed to be the cause of disease in cattle.

Respectfully,
E. A. DE SCHWEINITZ,
Chemist, Biochemic Laboratory, B. A. I.

Dr. D. E. SALMON,
Chief of Bureau of Animal Industry.

CHEMICAL EXAMINATION OF CORNSTALKS PRESUMABLY THE CAUSE OF CORNSTALK DISEASE IN CATTLE.

By E. A. DE SCHWEINITZ.

In 1890 some cornstalks were brought to the laboratory for examination. These had been collected from a field in which a few cattle had died from the so-called cornstalk disease, and were supposed to be the cause of the fatal result. The leaves and stalks were covered with smut. This was carefully separated before the examinations were made. An extract from the stalks gave reactions which would indicate the presence of cholin. Although this has been found in a number of plants, so far as I know it has never been noted in this connection, nor was the quantity present sufficient to have produced any poisonous effects in animals feeding on the stalks.

In 1892 and 1893 Dr. Moore undertook a more extended study of the cornstalk disease in the field, and collected and forwarded for study several packages of stalks. These were examined by Mr. Emery, under my direction. The dried powdered stalks were first extracted with petroleum spirits, and the residue then extracted with alcohol acidified with 3 per cent hydrochloric acid. This extract upon evaporation was slightly charred, but the water extract of the residue gave a yellowish green precipitate with phosphomolybdic acid. The water solution was then made alkaline with soda and extracted with ether, but no reaction for alkaloids could be detected. The solution made acid and again shaken with ether gave a small quantity of a brown, oily substance. The latter, however, gave no alkaloid reactions and failed to produce any effect upon guinea pigs when fed to them in considerable quantity. The petroleum extract was also free from poisonous principles. Another lot of stalks, leaves, and a few ears of corn collected from a portion of a field where animals had become ill were examined. The stalks, husks, and kernels of this lot were all examined for alkaloids or poisonous principles, but failed to show any reactions, with the exception of a slight precipitate with phosphomolybdic acid.

Some corn smut carefully separated from the above leaves and stalks was next extracted with alcohol acidified with hydrochloric acid. The extract was concentrated, neutralized with soda, and shaken with ether. The ether extract was evaporated spontaneously and the residue taken up with acidified water. The following reactions were obtained in this solution:

Iodine in potassium iodide, a red-brown amorphous precipitate.
Phosphomolybdic acid, a dirty yellow amorphous precipitate.
Mayer's solution, a whitish amorphous precipitate.

The solution was then neutralized with soda extracted with ether, and the latter solution concentrated over sulphuric acid in vacuo. A very small quantity of an oily nonpoisonous residue was obtained.

Some of the leaves sent in by Dr. Moore were covered with dark spots due to a parasitic disease. The diseased portions were carefully cut out and examined for alkaloids or poisonous principles.

The oily residue from the smut and also that from this part of the stalks appeared to correspond to sclerotic acid, a principle which Dragendorff has extracted from ergot and which Parsons claims to have found also in ustilago (corn smut). The quantity, however, obtained from the stalk would not be enough to account for the fatal effects in cattle unless a peculiarly susceptible condition of the animal existed at the time, or the improbable supposition that an excessively large amount of the poison was contained in the leaves and stalks that the animal happened to eat.

An enlarged and badly congested liver was also forwarded by Dr. Moore. This had been taken from one of the animals that had died supposedly from the cornstalk disease.

The liver was extracted with 90 per cent alcohol plus 20 per cent tartaric acid and the filtrate was evaporated in a vacuum pan. The residue was extracted with 95 per cent alcohol, and after reevaporation the residue was extracted with water. The solution gave a slight precipitate with phosphomolybdic and phosphotungstic acids, but no other alkaloidal reagents. The solution was then concentrated and mercuric chloride added in excess. After standing several days a precipitate was thrown down. This was filtered off and decomposed with sulphuretted hydrogen. After the mercury had been separated the residual solution gave a white curdy precipitate with phosphomolybdic acid. The filtrate from the mercuric chloride precipitate gave no reaction with alkaloidal reagents. The solution which gave a precipitate with phosphomolybdic acid was made alkaline with sodium hydrate and extracted with ether and chloroform. The water solution of the residue from these solvents gave a slight precipitate with phosphomolybdic acid but with no other reagent. It is true that this is the only reagent that gives a distinctive precipitate with sclerotic acid, but the precipitate here could scarcely be supposed to be due to the presence of this substance. The results of these examinations must be considered negative in character so far as the presence of an active poisonous principle is concerned.

The determination of the amount of ash in the cornstalks showed in the dry matter 6.4 per cent, and an examination was made for possible excessive quantities of nitrates.

Recently cattle have been found poisoned from cornstalks containing an unusual amount of potassium nitrate (Bulletin No. 44, Kansas experiment station). In the stalks, however, in which this was found the potassium nitrate could be detected by the eye in large crystals, and also by the taste. The particular stalks had been grown under peculiar conditions near the barn, where excessive amounts of nitrates could be absorbed. Those that we examined contained only a minute trace of the nitrate, so that if in any of the instances the cornstalks were the cause of disease in the cattle, and our samples were fair specimens, the poisoning was not by this salt. It should be remembered, however, that in all our examinations of the stalks the quantity used was small and not proportional to the amount cattle would eat. An apparently nontoxic principle under peculiar conditions might produce disasterous results.

INDEX.

Page.

- *Aspergillus glaucus* 49
- Bacteria in affected animals 58
  - cultures in blood 58
  - diseased cornstalks 40, 41
- *Bacillus cloacæ* 45
  - description 46
  - in cornstalks 40, 48
    - soil 45
- *Bacillus coli communis* 21, 76
- *Bacillus fluorescens liquefaciens* 73
- Billings, Dr. F. S 10, 13, 57, 62
- Burrill, Prof. T. J 15, 39, 45, 59
- Calves inoculated with rabies 76
  - virus from cattle 76
    - dogs 83
- Cattle disease at Emmetsburg 86
- Chemical analyses 49
- Cholin, occurrence in cornstalks 88
- Corn blight or the Burrill disease of corn 39
  - effect of fertilizers 41
  - rabbits fed on affected cornstalks 42
- Cornstalk disease in cattle 9
  - conditions under which it occurs 50
  - economic importance 62
  - etiology 10, 59
  - geographic distribution 9
  - history 9
  - hogs fed upon carcasses of diseased cattle 28
  - investigations by Billings 13
    - Gamgee 10
    - Illinois Experiment Station 15
  - nature 9
  - outbreaks 20
  - pathology 12, 55
  - prevention 64
  - symptoms 9, 11, 53
- Cornstalks as food for cattle 10
  - cholin, occurrence 88
- Enteritis in cattle 60
- *Enzootic cerebritis* 49
- Farmers' Review, summary of reports of prevalence of cornstalk disease 63
- Gamgee, Prof. John, indorsement of smut theory 10
- Going, Dr., investigations 60
- Hay, Leon, investigations 41
- Horses in cornstalk fields 34

Page.
Impaction, cause of death in cattle fed on dry food 57, 60
Kilborne, Dr. F. L., investigations and experiments 20, 23, 30, 44, 47, 83
Letter of inquiry, object 16
Mayo, Dr. N. S., description of a disease in horses 49
    cattle poisoned, potassium nitrate 61
McClure, Professor, observations on dwarfed corn 41
Niles, Dr. W. B., assistance in post-mortem examinations 19, 23, 27, 59
Nocard, Professor, isolation of bacillus of bovine pneumonia 14
Potassium nitrate 61, 89
    cattle poisoned 61
    occurrence in cornstalks 61
Pneumonia, relation to cornstalk disease 57
Rabbits inoculated with cornstalk disease, immunity 39
    rabies, effect 73
        virus from cattle, experiments 73, 78
            dogs, results 83
Rabies, comparison with other diseases in cattle 71
    history of outbreak in cattle 71
    lesions 73
    symptoms 72
    in dogs 83
Russell, H. L., investigations of bacteria in living plant tissue 47
Sclerotic acid, occurrence 89
    tests 89
Schroeder, Dr. E. C., observation 64
Schweinitz, Dr. E. A. de, chemical examination of cornstalks 49, 88
Sheep in cornstalk fields 28
Smith, Dr. Theobald, inoculation experiments 45, 77
Stalker, Dr. M., investigations 19, 71, 86
Toxæmia maidis 9, 62
Ustilago maidis 10, 18, 47
    feeding cattle with 47
Wade, John, experiments on farm 71

◆